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RINGWORM INFECTION OF THE HANDS AND FEET: A CONSIDERATION OF ITS RECURRENCES

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(Read before the Medical and Surgical Association of the Southwest, at the fifteenth annual meeting, held in Phoenix, Ariz., Nov. 7 to 9, 1929.)

The superficial fungus infections of the hands and feet, commonly known as ringworm, eczematoid ringworm, or dermatophytosis, are becoming more and more prevalent, due probably in large measure to the increasing use of gymnasiums, club houses, and public swimming pools. The prevalence of the infection is indicated by a recent survey of freshman students in the University of California. Legge, Bonar, and Templeton[1] found, among 3100 freshman entrants that 53.3 per cent of the men and 15.3 per cent of the women students were infected with ringworm of the feet. When one considers that at least a small per cent of those infected will be disabled for a time, one realizes the importance not only of getting the patient rid of the visible evidence of the disease and the annoying itching, but also of eradicating the dormant foci of fungi, and otherwise eliminating as far as possible the possibilities of recurrence.

It is not difficult in most cases for one familiar with the disease to clear up the gross signs of the infection and the subjective symptoms. After this the real problem begins. The difficulty of completely eliminating the fungus from the skin results in frequent recurrences of the eruption, and the neglect of outside sources of contamination means reinfection of the patient and spread of the disease to other susceptible persons.

At the present time a good proportion of physicians recognize, or at least suspect, this type of infection, and are able to treat the disease successfully up to a certain point, that point where the gross lesions disappear. Too often this is the end of treatment—until the recurrence, which is very likely to come; so likely, in fact, that someone has said it is necessary to burn the clothes and boil the patient in order to cure the disease permanently. If we will be thorough in our treatment and attend to the causes of recurrences our view of ringworm of the hands and feet becomes much more optimistic.

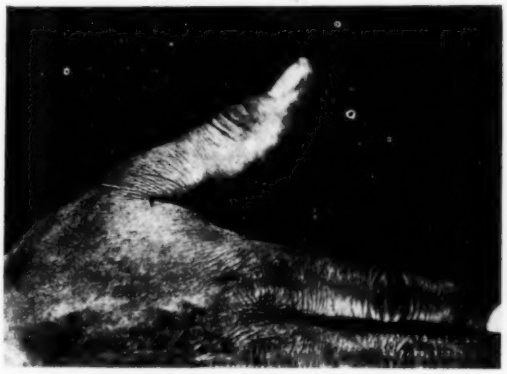

Fig. 1. Ringworm. Non-itching epidermal collar. Scales from these are often loaded with mycelia.

One of the most common causes of recurrence is a low grade infection which causes no pruritus and no noticeable lesion except an occasional small scale or circular collar of epidermis (Fig. 1). This may be the remnant of a more severe infection which treatment almost cured, or the infection may have been from thhe start a very mild one. These cases are often not treated, hardly even noticed by the patient, but are sources of in-

fection to others and cause repeated recurrences in the patient. Even without treatment they will often disappear, but the organisms are still present and will cause trouble at some time when conditions are favorable to their growth. If the physician will be on the lookout for this type of lesion in all his patients, he will discover many mild cases of ringworm which may be attended to before they cause annoyance. These lesions are most often found on the fingers, toes and palms. Between the toes there may be a very slight scaliness or an area of macerated skin which had never been noticed by the patient. (Fig. 2).

Fig. 2. Ringworm. A slight scaling between the toes which often passes unnoticed.

Another often neglected lesion is the mycotic onychia or infection of the nail. This may be present as a marked thickening of the nail with piling up of epidermis beneath the free border, or there may be a slight involvement with only discoloration (Fig. 3). The latter may easily be overlooked. If ringworm of the hands and feet is treated and the nail infection ignored this serves as a source of reinfection of the skin. Williams and Barthel[2] of the New York Skin and Cancer Hospital have found nail infection to be frequent and a common source of recurrence of the skin lesions.

It is my opinion that there are a great many ringworm carriers, individuals who habitually harbor the organisms without developing active infection themselves. These persons I believe to be foci for the spread of the disease to others, and in time they will probably develop the disease themselves in an active form. In my work at the El Paso City-County Clinic I decided to examine a

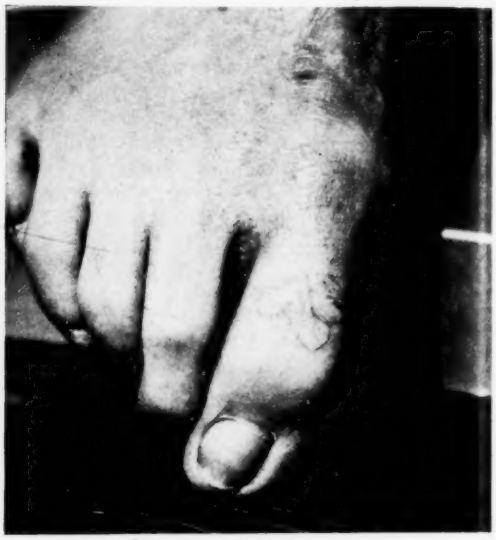

Fig. 3. Epidermal collar at edge of first interdigital space, and slight involvement of nail. Fungi found in scale. No subjective symptoms.

number of apparently normal toes, if such could be found, for the presence of fungi. The difficulty of finding normal toes among these clinic patients is considerable. Most of the patients are Mexican laborers and their families. Their personal hygiene is of an extremely low order, and active ringworm of the toes is almost the rule. After much searching ten patients were found who, as far as the eye could tell, had no infection. These had had no itching, and there were no scaling, maceration, or other signs of infection. Those patients who had even the slightest scaling were not used. Epidermal scrapings were taken from the fourth interdigital space of the foot and mounted in 30 per cent potassium hydroxide for microscopic examination. Of these ten apparently normal feet, five were negative, one showed an abundance of yeast-like organisms, and four showed branching mycelia resembling those found in active ringworm of the extremities. No attempt was made to classify the organisms further. While the organisms are existing here as saprophytes, I believe these cases are potentially active ringworm infections, depending on proper conditions for growth and infection.

I have so far considered the causes of recurrence about the patient himself. There are other important reasons why ringworm

recurs. Various articles with which infected parts of the patient's skin have come in contact may hold small scales containing fungi. These organisms are notoriously long-lived. In order to have some first-hand information concerning their longevity, I obtained some thin scales from an active foot infection, placed them between two clean glass slides and kept them in a dry place for three months. At the end of that time the laboratory succeeded in growing both a tinea and a yeast colony on Sabouraud's medium. It is very likely that the organisms will live in detached scales for much longer. Therefore, such articles as gloves, sox, bath mats, slippers, rugs, bath room floors, broom handles, tools, automobile steering wheels and brake handles, and others which might be contaminated should receive attention. Some of these can be sterilized in formaldehyde vapour; others will have to depend on soap and water; some that cannot be sterilized should be discarded. Those articles which can be, should be boiled.

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RAGWEED HAY-FEVER IN THE SALT RIVER VALLEY—SECOND NOTE

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(Read before the Maricopa County Medical Society, at Phoenix, Ariz., at a regular bi-monthly meeting.)

Last year I called attention to certain changes which are taking place in the distribution of pollen disease in central Arizona. The most important of these changes is the development of a definite autumnal hay-fever season caused by the pollen of the slender false ragweed, Franseria tenuifolia.

This year (1929) I returned early in October and found a number of patients seeking relief from autumnal hay-fever. Two-thirds of these patients had been treated with an extract of the pollen of Bermuda grass in April and May, and had obtained complete relief. They had been free from symptoms during the summer, receiving no further treatment. At some time in September, the exact date varying from the fifth to the twenty-fifth, their hay-fever had recurred. In most cases the symptoms were severe, several having asthma when they first reported for treatment. The records of those who had been tested recently showed some sensitization to Franseria, and in all of these recurrent cases there was a positive reaction to this pollen by the intradermal test.

The other group, about one-third of the total number, were not Bermuda-sensitive. Several had required treatment last spring, or in earlier years, with the pollen of the rabbit bush, Franseria deltoidea, during the short period of its pollination in March. Some had been treated last fall with valley ragweed. Their autumnal hay-fever was similar in onset and in severity to that experienced by the group who were also sensitized to Bermuda grass.

It was interesting to observe that less than half of the patients of each group had a history of ragweed hay-fever before coming to the Valley. The others, including several children born in Arizona, must have developed sensitization here.

A hurried and incomplete survey of the Valley pollen plants showed that the slender false ragweed was in full bloom. The area infested by this plant was much greater than in former years. There were found, at the edges of the city, whole fields of it. Patches of it occurred along the roadsides, much as the true ragweed grows in the East. These were found well beyond the limits of irrigation; in fact, I collected specimens as far north as Hillside.

The plants showed extraordinary variability in size and in conformation, possibly as the result of differences in soil and in moisture. The Valley specimens were identified by Mr. Robert H. Peebles, a botanist in the Government Service, as Franseria tenuifolia. This agrees with identifications made in earlier years by Dean Thornber and by Professor Hall. There is more or less western ragweed (Ambrosia psilostachya) in this region, and it grows, in some places, along with the franserias, and pollinates about the same time. I have not been able to demonstrate that there is any true short ragweed in the Valley; some observers think that there is. It is not of present importance, for undoubtedly the false ragweed is the dominant species, and it caused this year's autumnal hay-fever. It grew vigorously and went to seed; we may look for still more of it later. Patches in different parts of the Valley varied in their degree of maturity, which probably explains the irregularity of the dates of onset; but they all went to seed about the last of October.

The treatment was simple. The intradermal test of 0.02 c.c. of the 1:5000 dilution of valley ragweed pollen extract was the initial dose. Daily doses, increased as rapidly as the patient's tolerance allowed, were giv-

en by the intradermal method. Relief occurred in all cases, and usually it was prompt; but a few patients had to be protected from the pollen for a few nights, by a glycerin mask, by dampened sheets hung over the windows of their rooms, or some such device.

The pollen extract used for tests and treatment was made from naturally ripened pollen, collected from wild plants growing in the Valley. This pollen was carefully freed from dust and plant detritus, and its fat was removed, before extraction. It has been my experience that such preparation is necessary in making an extract that is stable and of standard potency.

A few of the Bermuda-sensitized patients received some doses of that pollen, along with the ragweed, but the Bermuda was soon discontinued. Obviously, it was not needed. If these people had gone comfortably through the summer they could hardly be suffering from the dwindling quantity of Bermuda pollen in the air in the fall.

SUMMARY

Autumnal hay-fever caused by the pollen of the slender false ragweed, Franseria tenuifolia, is present in the Salt River Valley. The season is fairly definite, beginning about the middle of September and ending late in October.

The number of persons affected is not great, but is increasing yearly. The offending plant is extending its distribution in vacant lots and along roadsides.

Persons who have had hagweed hay-fever from true ragweed, before coming to Arizona, are liable to be affected by the false ragweed; but in more than half the cases observed the sensitization developed in this region.

Patients who, after treatment with Bermuda grass pollen extract in the spring, have been comfortable all summer but have recurrence of symptoms in the fall, are usually sensitized to false ragweed. They then need treatment, not with Bermuda grass pollen extract, but with that of the false ragweed.

Sufferers from autumnal hay-fever in the Phoenix neighborhood were easily relieved during the attack by the intradermal injection of the appropriate pollen extract.

I am indebted to Mary C. Phillips, A. B., for valuable assistance in mapping the distribution and collecting the pollen of the Valley ragweed.

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SOME DETAILS IN THE TREATMENT OF HEART DISEASE

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(Read before the Medical and Surgical Association of the Southwest, at the fifteenth Annual Session, held at Phoenix, Ariz., Nov. 7 to 9, 1929.)

To those who are particularly interested in treatment of patients complaining of symptoms referable to the heart, details of diagnosis are of little importance. The arteriosclerotic patients may respond differently from rheumatic ones, but the plan of treatment in both is the same, and in either the details must be altered to fit the symptomatic, not etiologic, classification. When heart failure occurs, the chance of improving the condition by medication is greater if the patient has recently developed a wholly irregular pulse, but the treatment of auricular fibrillation is not a different chapter from the treatment of heart failure without arrhythmia. However, one etiologic factor, hyperthyroidism, will be sought because of its importance in therapy. Any cardiac patient, without obvious valve lesion, who is alert, with soft, pigmented, moist skin, slight flush, and rapid pulse or arrhythmia or high pulse pressure, should be suspected of hyperthyroidism, until it is disproved, since proper surgical intervention can relieve most of the thyroid group of cardiac patients of present distress and reduce the risk of future invalidism.

In treating a patient with congestive type of heart failure, it is usually wise to begin with a period of complete rest, and in the severe cases this is absolutely necessary. Strict bed-rest is preferable, but it is oftener more exertion for the patient to use a bed pan than a bedside commode, and in patient with prostatic disease, acute retention may result from enforcing continuous confinement to bed. The position of the patient in bed is very important, since many of them are comfortable only when sitting up. The hospital cardiac beds are convenient for this, but it is possible, by having the patient's feet against the foot of the bed, a pillow under the knees, and a back rest of pillows and boxes extending to the head of the bed to furnish firm and comfortable support even for the patient at home. If the patient is merely propped up on pillows in the center of the bed, most of his time is spent in tiresome sliding and climbing.

The diet during the first day or so may be light or liquid, but as soon as possible, unless the patient is overweight, he should be getting full diet, in several moderate-sized feedings, chiefly carbohydrates, with adequate

fresh fruit juice, dairy products and vegetables; greasy and "rich" items should be omitted. Even in obese patients, a carbohydrate diet with sufficient vitamine content should be provided. As little salt as possible should be given with the diet. The fluid intake need not be markedly restricted, but no water should be left at the bedside or within sight of the patient, so as to avoid stimulation to excess fluid intake. In thirsty mouth-breathers, it may be necessary to allow only a limited intake, with cracked ice to suck every twenty minutes. In moist, cool weather, thirty-five to fifty ounces daily total liquid intake is quite comfortable, but in dry, hot weather this may cause distress. A record of intake and output of fluid, and frequent weighing of the patient, is useful in controlling therapy.

Digitalis is the one drug which is of importance solely because of its action on the circulation. It has several distinct effects upon the circulation, as well as toxic actions which are well known. It increases vagus tone, which tends to slow the sinus rate and the conduction from auricle to ventricle. It improves the efficiency of cardiac contraction so that less energy is required to pump a given amount of blood. It increases the speed and force of cardiac contraction, but only when the heart is dilated or failing. Also, it slightly diminishes the return flow to the heart by action on the peripheral circulation. In auricular fibrillation, the vagal and direct action on conduction from auricle to ventricle causes marked slowing of apex rate in most cases. When the beat is regular, digitalis may not produce any slowing. It rarely does so in fevers, anemias, and hyperthyroidism.

Digitalis should be given, sooner or later, to most cases of congestive failure. In fibrillation and when the condition is critical, it should be given at once; in other patients, one can estimate the value of the drug best by withholding it for a few days. The speed with which full effects are to be produced should vary with the seriousness of the need for the drug. In rapid fibrillators I should expect to secure full effects in one to three days; in patients who are not critically ill in three to six days. The average requirement is four drams of tincture or twenty-four grains of powdered leaf per 100 pounds, but the variation from patient to patient is close to twenty-five per cent in each direction. Give one-half of the required amount in one dose, in urgent cases one quarter more in six hours, and then three grains every six hours until the apex rate is slowed to eighty, or the patient has toxic symptoms such as diarrhea, coupled beats or nausea. If the patient has had abdominal pain and nausea previously, the digitalis nausea can usually be distinguished by the dizziness and headache which accompany it. In less acute cases the drug can be given in three grain doses three times a day for two days, twice a day for two or three days and three grains, or one-half dram, daily until one gets evidence of toxicity or full clinical effectiveness. If toxic symptoms occur, omit for a day or two and then resume the same sized doses at longer intervals, five days a week, or every other day, as needed to maintain the full effect.

Fibrillators who have done well on digitalis need to be rationed, with the largest dose they can take, for months or even years, and discontinuing may cause heart failure. When the heart is regular, digitalis can be discontinued only if it does not benefit the patient, or when he has returned to a stationary condition. It should be begun again, in full doses, if its withdrawal causes aggravation of symptoms. The cardiac must be educated in regulating digitalis as the diabetic is for insulin. When the patient has had digitalis before beginning treatment under your care, smaller doses may suffice to produce results, although in my experience daily doses of twenty to thirty drops from a dropper are equivalent to no medication. Either use powdered leaves, or tincture measured from a minim glass, if you wish to get results. Even accurate medicine droppers give from 120 to 300 drops instead of sixty to the dram. Do not divide your dose up, but give it once a day for maintenance. It is cheaper, if you use powdered leaf, surer if you use tincture, to give only one dose a day. Some patients prefer bitter taste to the pill sensation, but if possible, I use powdered leaves. There is no need for purified forms or extracts when giving the tincture by rectum, as is sometimes necessary in fevers, after operation, or with severe gastric disturbances.

Next to digitalis, morphine is our most needed drug. It is invaluable during the first day or two of treatment of severe decompensation, as well as for acute attacks of pulmonary edema or of severe pain in coronary arterial occlusion. By greatly diminishing activity and anxiety, it rests the patient and decreases the work of the heart, but it makes him more cyanotic and his breathing more irregular. It is rarely wise to use more than two one-fourth grain doses of morphine per day in congestive failure, but more may be used after coronary occlusion. Caffeine is of value as a respiratory stimulant, when the patient is being disturbed by the overbreathing periods of

Cheyne-Stokes respiration, and as a general stimulant in collapse. It should be given intramuscularly in four to ten grain doses of the sodium salicylate or benzoate. If these have to be repeated frequently, they may produce or exaggerate as "cardiac psychosis" or manic state. Camphor, carbiazol and coramine are worthy of trial as stimulants, although they are rarely superior to, and often less effective than, full doses of caffeine. Digitalis action may be reinforced for a few hours by intravenous injection of ten cc. of 10 per cent calcium chloride, but this is not often indicated.

Diuretics are often important, either as support to digitalis, or for use after it has done its best, whether successfully or not. Theocine by mouth, seven grain doses two to three times in one day, and repeated not oftener than every third day, gives a very satisfactory diuresis and is usually quite as effective as its expensive cousin, euphyllin, and more so than mercurial diuretics, in many patients. Occasionally when theocine (or the less effective but better tolerated theobromine, in ten grain doses thrice daily) seems unsatisfactory, we see good diuresis from the mercurial diuretics, salyrgan or novasurol, given intravenously in one or two ccm. doses, one to three days apart. The mercurial diuretics are most effective if the patients are kept acidotic by large doses (six to ten grams daily, usually divided into eight hourly doses) of ammonium chloride or nitrate, or calcium chloride. These drugs alone may produce a moderate diureses. I prefer ammonium chloride, ten grains every hour for ten to fourteen doses daily, but all of these drugs cause gastric distress. The mercurial diuretics must be avoided if diarrhea is present or if they produce bowel symptoms.

Last, but not least, we come to the mechanical procedures which are so valuable in heart failure. First, is venesection, which is unapproached for giving quick relief in cases where orthopnea, pulmonary edema, or swollen liver are outstanding features. It is useful during the first few hours even when fibrillation is present and relief within a few days, under digitalis, seems certain, more useful in failure without irregular pulse, or in patients who respond poorly to digitalis and diuretics. Most diuretics work better after a bleeding. The easiest and most successful technic is to use a twelve to sixteen gauge needle with eight to ten inches of tubing applied over the butt of the needle, not with an adapter. Never use a tourniquet, with its uncertain pressure, but keep a blood pressure cuff at sixty mm. mercury or on up to ten mm. below diastolic pressure during the venesection to secure the freest flow. The rubber tube should hang straight down into a graduate, and 500 to 800 c.c. (eighteen to twenty-four ozs.) should be withdrawn to produce a satisfacory fall in venous pressure.

It is wise to slap the cardiac on the back every morning to see whether he has fluid in the chest and, if so, how much. When flatness reaches above midscapula, he usually should be tapped. In patients with fibrillation, recently come under your care, marked hydrothorax may take care of itself, but if it does not, much relief can be obtained by removing fluid. Some cardiacs require repeated abdominal paracentesis, and this also is a procedure which often makes possible a good result from diuretics. I prefer mid-line taps, with a gallbladder trocar and attached rubber tube, but puncture above the left iliac crest is better borne by some individuals. Occasionally, in patients with massive edema resistant to every form of therapy, hanging the legs out of bed and allowing fluid to drain from two to four skin incisions, two to three inches long at the level of the malleoli, will turn the scale and initiate diuresis as well as directly reducing the amount of stored fluid.

If patients fail to gain under rest, proper diet and medication as suggested above, one is reduced to keeping up the battle, maintaining strength by proper care of the bowels, relief of indigestion and sedatives to produce sleep. Spontaneous improvement may begin after weeks of discouraging progress. I prefer using no powerful cathartics, although some men believe in calomel for resistant cases of anasarca. Small doses of cascara, phenolphthalien, or mild morning saline cathartic are usually adequate to avoid hard stools, and produce little abdominal distress with attendant fear of eating. Paraldehyde by rectum, or with ice by mouth, chloral, veronal and phenobarbital are all worth trial to produce comfortable sleep.

The use of quinidine to effect restoration of regular rhythm in auricular fibrillation, is best confined to patients who have had this disorder for only a few days, or who have been well prepared by digitalis, so as to regain as much cardiac reserve as possible. While the risk of accident is small in patients who are ambulatory and comfortable, the improvement produced is variable and sometimes negligible if the patient had a slow pulse under digitalis. Recurrence of fibrillation is not uncommon, even when patients take quinidine daily to maintain a regular rhythm, and decompensation may occur when fibrillation returns. Usually the drug is giv-

en in capsules, beginning with three grains three or four times daily, and increasing by one and a half grains per dose each day until the patient is distressed (usually this will require nine to eleven grains four times daily) or until regular rhythm occurs. The latter is often preceded by a few huors of tachycardia which is sometimes alarming.

When patients begin to improve, one is faced with the problem of exercise and, later, return to work. When actual heart failure has occurred, the patient should be eating well, sleeping well and free from dyspnea for five to ten days before allowing him to move about his room. He should begin his conva'escence by sitting in a chair for fifteen minutes two or three times a day, increasing this so that within one or two weeks he is sitting up for all meals, lying down for one-half to two hours after each, and walking on the level or sitting up as much as he desires outside of these fixed periods, with ten hours bed rest at night. One should clear up any serious sources of future trouble, such as abscessed teeth, pelvic or prostatic disease, before convalescence is over.

The recovery of function in patients with fibrillation, valvular lesions, or recent coronary accidents, is sometimes more rapid and complete than in those with less serious heart disease complicated by poor physical condition, obesity or a sensitive nervous system. However well they feel during convalescence, they should be urged to avoid fast climbing, lifting, and all competitive athletic events. Recurrence of edema of the ankles, cough, or nocturnal dyspnea, indicates need for more rest. Prolonged exposure, fatigue, exercise after meals, heavy meals alone, and intercurrent infections, are likely to bring out symptoms or lead to a serious relapse. Sexual intercourse should be avoided at times when the patient is tired and the act likely to be prolonged. I believe cardiacs are better when they have regular mild exercise, with rest after meals, but of course they must never push themselves if exercise causes distress. They should maintain their weight at, or slightly below, the "ideal weight" for age, sex, and height.

Another group of therapeutic problems which deserve mention is centered about the border-line cases. First, those with irregular, or enlarged hearts, with hypertension, or with definite diastolic murmurs, but with no distress on exertion or other symptoms of heart disease. For these no digitalis is needed but advice to avoid competitive sports and undue fatigue. Often this advice is ignored without evidence of damage and the first break in compensation comes as a result of upper respiratory infection. Hence, tonsillectomy may be of some value in selected younger patients with such troubles. In the next class for treatment are the individuals with many symptoms but no physical evidence of heart disease or a cause for heart disease, and those with some physical evidence of heart damage who have had only mild symptoms of functional insufficiency. The patients with angina pectoris may also be placed in this class. These patients sometimes are helped by an initial brief period of bed rest, followed by the same regime of avoiding exertion but obtaining regular exercise, which I have outlined for those convalescent from congestive failure. Exercise may be increased only very slowly in some of the patients, particularly those without physical evidence of heart disease for whom the diagnosis of "poor training," or "heart tire," "effort syndrome," etc., may be applicable. The slower the recovery of function, the more important is mid-day rest, avoidance of late hours and heavy evening meals in future life. Obesity, and chronic infection of mouth, tonsils, or elsewhere (notably pulmonary tuberculosis, presumably inactive) may be important complications in this group. Drugs are of little value, except sedatives and those with psychic appeal. Arsenic and digitalis may act in the latter fashion. Whether the patient actually has early heart disease, or not, is unimportant in therapy, for the treatment is based on symptoms, and emphasis on heart disease may introduce a psychic obstacle to recovery. Patients of this type who believe they are likely to die soon from heart disease will either neglect themselves or, by too great caution, produce actual damage from failure to maintain good physical tone.

It is apparent that the heart is somewhat damaged by all acute infections, and it is put to an unusual strain by operation and blood loss. However, the role of true heart damage in infections and operation has been greatly exaggerated. Most patients with heart disease, even those who have had, or still have, some evidence of failure, bear operation well. Yet many patients with normal hearts show tachycardia, cyanosis and fall of blood pressure after prolonged operation. These disturbances are peripheral in origin, and, while full doses of digitalis by rectum, or strophanthus by vein, may help to raise blood pressure, the safe, wise and rapid method of treatment is saline infusion or blood transfusion, perhaps also some overbreathing with oxygen-carbon dioxide mixtures. Later on after operation, paralytic ileus, dilation of the stomach, or pulmonary infarction may cause symptoms suggestive of heart failure. These symptoms are rarely cardiac in

origin, and respond poorly to cardiac medication, which often is used instead of appropriate measures. Similarly, in infections, peripheral circulatory collapse is not uncommon, and is best treated by transfusion and repeated small doses of epinephrine. In pneumonia, particularly in elderly people, full digitalis dosage or venesection has often seemed to save life. Probably, even in these cases, proper oxygen administration would have been more effective.

In summing up, let me urge that we abandon the simile chosen by the illustrious Corvisart, who likened the cardiac to Virgil's soldier "fixed in whose side was the lethal shaft." Heart disease is not often immediately or progressively fatal, but goes by remissions and exacerbations. During the latter, our best measures may prove unavailing and still, in some cases nature will gradually restore the balance. We must never state a definite time of survival for a patient even when things seem most desperate, but maintain in ourselves and in our patients a feeling of cautious optimism.

ABDOMINAL SYMPTOMS DUE TO CARDIOVASCULAR DISEASE
A DISCUSSION OF POSTMORTEM CASES

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(Read before the Medical and Surgical Association of the Southwest, at the fifteenth annual meeting held at Phoenix, Arizona, Nov. 7 to 9, 1929.)

That heart failure may cause digestive disorders is well known. The role of arterial disease in abdominal symptomatology is not so much appreciated.

In the first place, it must be understood that practically any blood vessel in the body may become painful when an adequate stimulus is applied. Nearly all of the hollow viscera become painful when over-contracted, over-distended, stretched, or when irritated by certain chemicals. Injection of warm water under pressure into the gall ducts causes suffering; barium chloride injected into the lumen of an artery causes spasm of the vessel walls and pain.

A sclerotic artery cannot contract, relax or bend easily, and for these reasons may, more readliy than the normal artery, become irritable and hypersensitive, and thus may become a focus for afferent impulses, just as much as a gastric ulcer or an inflamed appendix. Sudden occlusion of any artery is generally very painful.

CASE 1.

A man aged 65 years was referred to me because of nocturnal dyspnea, high blood pressure, and loss of his usual vigor. In a few days his heart began to fibrillate, and this was followed by severe abdominal pain, colicky in nature, and of indefinite localization. There was marked obstipation and no passage of gas for two days.

Finally four ounces of castor oil caused bowel movements. However, the violent pains continued, and became localized over the left kidney and ureter, and percussion over the kidney was painful. The pains grew worse and the patient rolled in agony. The abdomen became distended, rigid, and tender everywhere. The bowel movements now contained mucus and considerable blood. As there was doubt about the diagnosis, it was decided to explore the abdomen. Just before making his incision, the surgeon remarked: "I feel that we are going to find a ruptured appendix." My own diagnosis had shifted from stone in the kidney to mesenteric thrombosis. As soon as the incision was made, black gangrenous bowel protruded. Mesenteric thrombosis was found at autopsy.

This case brings out two points: (1) The mesenteric arteries are highly sensitive when suddenly occluded; and (2) the pain and other symptoms closely simulate common surgical conditions. The persistent obstipation was probably due to reflex ileus. The presence of auricular fibrillation suggests the possibility of embolism from a clot in the left auricular appendage.

CASE 2.

A physician aged 50 years suffered from high blood pressure, and angina of effort for 10 years. January 28, 1928, he had a typical attack of cardiac infarct. March 27, there was a second infarction followed by several small hemoptyses.

A few days later there was severe pain in the left hypochondrium, which radiated downward and suggested stone in the kidney or renal infarct. There was no blood in the urine. There was also pain in the region of the spleen, which was thought to be possibly due to lung infarct. The abdominal pain became general, the abdomen tightly distended and tender everywhere. There was complete obstipation. No gas was passed for four days, in spite of the most skillful treatment. Finally, fecal matter returned with enemas and peristalsis was reestablished. There was no blood or mucus in the passages. April 8, 1928, he suffered a sudden complete right-sided paralysis with aphasia, all of which persisted until his death, April 20, 1929.

At post-mortem the heart showed infarct of the left ventricle and both coronaries were markedly sclerosed. The thoracic aorta was only moderately atheromatous, but the abdominal aorta was markedly atheromatous, with many large calcified patches, and all of its branches were thick-walled and tortuous. The spleen was small and scarred by old infarcts and the left kidney had a scar extending its whole length, greatly damaging the cortex. There was no pathology in the gallbladder or the digestive tract.

In this case we have marked vascular pathology in the abdomen, extensive arteriosclerosis, and splenic and kidney infarction. These findings, I think, readily account for all of the abdominal symptoms presented. The spleen and kidney are quite plentifully supplied with sensory nerves going direct to the cord, and not differing in any way anatomically from the cerebrospinal sensory nerves.

This is true of all afferent nerves from the abdominal viscera and the blood vessels ev-

erywhere. Afferent impulses, from what was probably embolism in the splenic and renal arteries, caused the intestinal ileus, and, by exciting vascular spasm, caused the abdominal pain.

The question of surgical intervention might have been considered in this case. So far, in our autopsies on cases of cardiac infarct, we have found no acute surgical pathology in the abdomen. We have found a quiescent gallbladder with stone, a number of times.

In about one-fourth of all cases of coronary sclerosis, there is sclerosis of the abdominal arteries. In cases of arterial hypertension, the arterioles are sclerosed in the pancreas in forty-nine per cent, in the liver in thirty per cent, in the spleen in sixty-six per cent, and in the kidney in over ninety per cent.

CASE 3

An American, aged 40 years was seen in December 1928, during an attack of influenza from which he died. Some 15 years before, he suffered a very acute attack of abdominal pain, during which he rolled on the floor in agony. A surgeon who saw him thought that he had appendicitis and wanted to operate. This was refused. He gradually got better, but continued to suffer quite constantly with gas in the stomach and pain that came on from a few minutes to two hours after eating, sometimes going to his left shoulder and arm. At these times moving his arm made the pain worse. His gastric suffering was so severe and prolonged that it was believed that he had ulcer of the stomach or some other grave digestive ailment. For this reason a post mortem was requested by the family.

At postmortem the entire digestive tract was carefully examined, and no lesion found: no ulcer, no appendicitis, no diseased gallbladder. The left ventricle of the heart was much enlarged and there was marked aneurysmal dilation of the apex. Over this area, the heart wall was very thin and composed mostly of scar tissue. The anterior descending branch of the left coronary was sclerosed and completely obstructed. The aorta was markedly sclerotic in both its thoracic and abdominal portions, and the orifices of the celiac and mesenteric arteries narrowed by atheroma.

Were his abdominal symptoms due to syphilis? I think not. Dr. W. W. Waite could find no evidence of syphilis in the aorta. (It should have been mentioned that there had been a faintly positive Wassermann reaction). The coronary arteries were atheromatous and not scarred at the orifice as we find in the coronary obstruction of syphilis. In the absence of confirming signs, a doubtful Wassermann is of little significance. We have had several cases of early coronary sclerosis. We are led to very strongly suspect that what was considered an attack of acute appendicitis was really acute coronary obstruction and that the subsequent abdominal symptoms were due to arteriosclerosis and circulatory disturbances in the abdomen. When heart symptoms are present in such a case, it is common to attribute the disturbance to indigestion or gas in the stomach. In the presence of general arteriosclerosis, this is, more often than not, a case of putting the cart before the horse.

On the morning of May 19, 1923, Mr. A. C., aged 65, seemed quite well. After breakfast, he started to cross the street but returned at once because of violent pain in the stomach, that went up into his chest, and he felt that he must die. He coughed violently and began to vomit blood, containing quite large clots. His physician thought he must have cancer or ulcer of the stomach. The pain continued for two days, and was followed by symptoms of heart failure.

In 1902 he was thought to have stone in the kidney and operation was contemplated; but the pain shifted to the other side and the diagnosis was in doubt. During 1923, he had pain in his legs, especially at night, which was relieved by heat. His blood pressure in recent years was high. After his first attack in May, 1923, his stomach continued to give him a great deal of trouble, but a complete examination by a competent gastro-enterologist disclosed no cause for his troubles in the digestive tract.

When he came under my care in August 1923, he was edematous, with tender, swollen liver, dyspnea, cyanosis, and nocturnal asthma. The heart was enlarged and there was a systolic murmur at the apex. He made a partial recovery, but continued to suffer with digestive symptoms and anginal pains on effort. He died January 26, 1928, from gradual heart failure. I had made a diagnosis of cardiac infarct in 1923. Postmortem showed cirrhosis of the liver. The stomach showed a thickened, congested, red mucosa. There were no ulcers nor diseased gallbladder. The kidneys were in fair condition. Both coronaries were markedly sclerosed. The anterior descending branch of the left coronary was obliterated by an old thrombus, but a fairly good collateral circulation had been established. The left apex was very thin and made up of scar tissue.

Was the gastric hemorrhage due to cirrhosis of the liver? This is not probable because, during the subsequent five years of his life, there should have been more hemorrhages. Gastric ulcer is excluded by examination and by postmortem. Fifteen per cent of gastric ulcers in those past middle life are said to be due to arteriosclerosis. With a thrombosed coronary, we may believe that there was also thrombosis in some small gastric artery, causing necrosis and erosion, which was the source of the bleeding and hemotemesis. We have had two other cases of gastric hemorrhage associated with cardiac infarct. The postmortem confirms the opinion that in this case the digestive symptoms were of cardio-vascular origin. The vague pains ascribed to the kidney may also have been due to arteriosclerosis.

CASE 5

A highly intelligent surgeon 55 years of age had suffered for many years with high blood pressure, indigestion, gas on the stomach and what he believed to be attacks of gallstone colic. On Christmas day, 1924, he ate heavily and that night had sudden dyspnea, vomiting, violent pain in the epigastrium, going up under the sternum and to both shoulders. He attributed his trouble to indigestion and rheu-

matism, and refused to go to bed. He died suddenly, January 25, 1925.

Autopsy revealed thrombotic occlusion of the descending branch of the left coronary artery, aneurysm of the apex and rupture of the interventricular septum. The gallbladder was normal and no pathology was found in the digestive tract.

A so-called gastric or gallbladder pain that is accompanied by pain in the left shoulder and arm often means coronary sclerosis, though abdominal lesions may be present at the same time. Pain due to arterial spasm is sometimes so similar to the pain of gallstone colic or duodenal ulcer as to be very difficult to differentiate. In fact, the pain of ulcer in the digestive tract is thought by some to be due to chemical irritation of blood vessels lying in the submucosa. In hemorrhagic pancreatitis, which sometimes comes into question in the abdominal symptoms of cardiac infarct, there is really a vascular lesion. Many of the sensory nerves of the pancreas go to the pancreatic arteries and, with arteriosclerosis in that area, an irritable focus could well give rise to the syndrome of pancreatitis, even without hemorrhage. However, a good deal of confusion is due to poor observation, for, when patients are asked to point out the location of their stomach pain, they often put their hands on the upper sternum.

CASE 6

A young lady of 22 years was seen Feb. 26, 1926. She had been sent to a surgeon for operation for gastric ulcer, but x-ray showed an enlarged heart and she was sent to me.

During 1924 she had had severe attacks with vomiting and pain in the stomach and hepatic areas. At that time gallstone colic had been diagnosed. At times she became bedfast. Exercise became difficult on account of dyspnea.

While under my observation, she continued to suffer with pain in the stomach and vomiting, and at times with pain in the shoulders and arms. The pulse was feeble and slow, but the blood pressure was high. The liver was swollen and very tender. The heart was very large. No murmurs were present. She finally developed general edema and died suddenly, April 15, 1926. My antemortem diganosis was congenital heart disease.

At postmortem the heart was found very much dilated, especially the left ventricle, which was thin and diffusely fibrosed. The aorta and its branches were thin, delicate and very narrow. The gallbladder was normal and no ulcers were found in the stomach or duodenum. The mucosa of the stomach was congested, thickened, and marked with punctate hemorrhages. The postmortem diagnosis was congenital vascular hypoplasia.

Quite evidently this case was cardiovascular from the beginning. Why should there have been such definite syndromes of gallstone colic and gastric ulcer?

The pain was made worse by food. I think the pain is explained by the increased work thrown upon the thin and narrow abdominal arteries in the attempt to furnish an adequate blood supply for digestion. The same difficulties were encountered in the coronaries, resulting in pain in the shoulders and arms. The fact that over-distention, as well as over-contraction, of a blood vessel will give rise to pain has already been mentioned.

In this case we have marked congenital vascular inferiority. Lesser defects might well cause similar symptoms.

It is said that fifteen per cent of all gastric and duodenal ulcers in the young are caused by local vascular spasm, perhaps similar to that of Raynaud's disease in the extremities. Localized vascular spasm in the splanchnic area may account for necrosis and hemorrhage and for many obscure abdominal pains.

Pain about the heart, upper chest, and in the arms is readily conceded to be due in many cases to sclerotic coronaries. We are not so quick to see that sclerotic arteries in the belly may cause even more widespread symptoms. The mechanism is just the same. Intermittent claudication in the leg offers a visible demonstration of what happens. Irritation in the sclerotic area of the artery by exercise, originates afferent sensory impulses to the nerve centers and excites sympathetic motor reflexes to the vessels lower down, causing spasticity and pain. The dorsalis pedis pulse disappears and there is blueness and coldness of the foot. In angina pectoris, strain on the sclerotic portion of the coronary artery, from exercise, over-eating, emotion, toxemia or infection, excites reflexes in the same way, with resulting spasm and pain in the non-diseased portion of the artery.

The same stimulus may result in vascular spasm and pain in the arm, because the arteries of the arm get their sympathetic nerve supply from the same cord area as the coronaries. In the belly, the same mechanism comes into play through sclerotic or other irritation in the celiac or mesenteric arteries, exciting viscero-motor reflexes by way of the splanchnic nerves. On account of the multiple contents of the belly, such reflexes have a widespread effect, as seen in the cases just discussed. The blood vessels go to every tissue in the abdomen and carry on their walls not only their own nerve supply but that to every organ in the abdomen. Aneurysm of the hepatic artery has been mistaken for gallstones. Hence, it is quite evident that afferent impulses from the vascular tissues in the belly may have much the same effect in causing symptoms as those from the other abdominal contents. A look at the heart and blood vessels, in cases with obscure abdominal symptoms, may avert serious mistakes.

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CORONARY CONCLUSIONS

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(Read before the Medical and Surgical Association of the Southwest, at the fifteenth annual session, held at Phoenix, Ariz., Nov. 7 to 9, 1929.)

Early in December of last year, a prominent physician of Yavapai County was found dead in bed. He had apparently died without a struggle, for his bed was quite undisturbed. He had always enjoyed excellent health and was noted for his unusual physique. Ten days prior to his death, while driving his car, he was suddenly stricken with a nagonizing pain over the precordia. The pain was so severe that he was forced to stop his car for a few minutes before he was able to start for home, where, upon his arrival, he was assisted from his car and a fellow physician summoned. He was nervous and restless and appeared to be in severe shock. His color was ashy, his skin cold and clammy. The pulse was feeble and the heart sounds unusually faint. He complained of a severe pain of extraordinary intensity over the precordia. Large doses of morphine were necessary before relief was obtained. Twenty-four hours later a friction rub was heard over the left chest in the region of the heart. The patient was feeling much better although a moderate amount of pain and distress remained.

At the end of a week he was somewhat improved, although he complained that he felt poorly and could not gain back his lost strength. He was out of bed and walking about the house, but was extraordinarily weak for a man of his unusual physique. The night before his death, he was feeling moderately well and was in good spirits. He retired in apparently good health at eleven o'clock and was found dead at six the following morning. A diagnosis of coronary thrombosis was made.

Since that time, four other fatal cases of coronary thrombosis have occurred in Prescott. I have personal knowledge of two of these cases. The other two were reported to me by my colleague, Dr. James H. Allen of Prescott, to whom I am indebted for permission to report them.

In this series of five cases the age of the patient varied from 43 years to 70 years with an average of 55.5 years. With one exception, all patients gave a history of previous attacks of precordial pain varying from a slight sense of discomfort, diagnosed by the patient as "intercostal neuralgia," to severe pain requiring large doses of morphine for relief. In no case was there demonstrable relation between exertion and onset of the attack. The duration of the fatal attack varied from thirty minutes to ten days. In one case, only, was there a friction rub heard.

Although the condition of thrombosis and occlusion of the coronary arteries has been known to pathologists for a long time, it has been only in recent years that clinicians have come to recognize it as a direct clinical entity which is, as Herrick says, "almost as distinct and clear-cut as a cerebral hemorrhage." It is by no means rare, and is probably the chief cause of sudden death in presumably healthy individuals.

PATHOLOGY

The cause of these thromboses seems fairly well established. Postmortem examinations, almost without exception, show chronic changes in the walls of the coronaries, usually in the anterior descending branch of the left coronary. These changes are described as patchy atheromatosis, calcareous plaques, narrowing at the point of bifurcation, arteriorsclerosis.

As a result of narrowing of the lumen, injury to the intima and sluggish blood flow, thrombus formation occurs. Gradual occlusion of a coronary may produce only a few symptoms, as collateral circulation may care for the affected area. Sudden occlusion of a large artery, however, usually produces sudden death, presumably through ventricular fibrillation. If death does not immediately ensue, infarction and anemic necrosis follow. If the softened area does not rupture within the next few days, recovery may occur, with either an aneurysmal bulging or plaques of fibrosis.

By far the great majority of cases occur in males between fifty and sixty. Many cases show chronic hypertension. Occupation, alcohol and tobacco are not determining factors. White has shown that syphilis plays a very minor role in the etiology of this con-

dition. Peripheral arteriosclerosis may or may not be present.

CLASSIFICATION

Not all cases of coronary thrombosis die suddenly. Some live for many months. Many cases are seen in which the symptoms are extremely mild. Herrick has grouped cases of coronary thrombosis according to clinical symptoms in the following manner:

"1. Cases of instantaneous death, a group graphically described by Krehl, in which there is no death struggle, the heart beat and breathing stopping at once.

"2. Cases of death within a few minutes or a few hours after the obstruction. These are the cases that are found dead, or clearly in the death agony, by the physician who is hastily summoned.

"3. Cases of severity in which, however, death is delayed for several hours, days or months, or recovery occurs.

"4. A group that may be assumed to exist, embracing cases with mild symptoms, for example, a slight precordial pain ordinarily not recognized, due to obstruction in the smallest branches of the arteries."

SYMPTOMATOLOGY

The commonest symptom is pain, usually coming on suddenly. Hammen has tabulated the symptoms in the following fashion:

1. The immediate symptoms associated with the occlusion; the original seizure.
   - A. Pain.
   - B. Shock.
     1. Prostation.
     2. Fall in blood pressure.
     3. Suppression of urine.
2. The symptoms associated with the myocardial damage, myocardial insufficiency.
   - A. Dyspnea.
   - B. Passive congestion.
     1. Cyanosis.
     2. Pulmonary edema.
     3. Enlarged liver.
     4. Albuminuria.
     5. Subcutaneous edema.
   - C. Cheyne-Stokes breathing.
   - D. Feeble cardiac impulse, faint heart sounds, gallop rhythm, murmurs, cardiac arrythmias.
3. The symptoms associated with myocardial infarct.
   - A. Fever and leukocytosis.
   - B. Pericarditis.
   - C. Embolic phenomena.
   - D. Cardiac aneurysm and rupture.
4. Additional symptoms.
   - A. Nauesa and vomiting.
   - B. Facies.
   - C. Vasomotor symptoms.
   - D. Nervous symptoms.

The electrocardiograph usually shows characteristic changes following coronary occlusion whether occurring spontaneously in man or produced artificially in experimental animals. There is an inversion of the T wave in leads I and II and a decrease in the amplitude of the QRS complex accompanied in many instances by notching, slurring or spreading of the latter. However, cases occur without the characteristic electrocardiographic changes, and as Warfield points out, the electrocardiographic evidence is valuable only when it is positive.

TYPICAL CASE: Patient number five may be cited as a typical case. A professional man, aged 43, of unusually regular habits, had complained of a series of minor, to moderately severe, pains in the precordial area for three or four months. Otherwise he was an unusually healthy and robust individual and had enjoyed good health for as long as he could remember. While doing light chores about the house, he was seized quite suddenly with a sharp agonizing pain over the lower sternum. The pain was infinitely more severe than any he had previously experienced. He was extremely restless and could not be quieted, but rolled about the bed complaining that the distress was so severe that he could not remain still. His face was ashen, his lips cyanotic, his skin cold and very wet. His pulse was almost imperceptible, thready, regular, rapid. He was nauseated, vomited. One-half grain of morphine was given over a period of an hour before partial relief was obtained. He then became somewhat quiet, but from time to time would toss himself nervously about the bed. The most striking feature of the case was the patient's ability to move about the bed when his heart sounds were scarcely audible, his pulse almost imperceptible and his systolic blood pressure only 60 mm. Hg. He appeared on the point of dissolution, but remained remarkably clear mentally and talked with the attending physicians. Within three hours, typical Cheyne-Stokes breathing developed, and the patient died five hours after the onset of the attack. The three attending physicians independently arrived at the same diagnosis.

DIFFERENTIAL DIAGNOSIS

In considering the differential diagnosis of coronary occlusion, one should think first of angina pectoris. How often death in angina pectoris is due to coronary obstruction is extremely hard to tell. Certainly more and more cases of angina pectoris are showing coronary obstruction at autopsy. However, true angina pectoris usually presents a picture differing in certain essential details from coronary occlusion. In angina, the attack is more apt to be induced by exertion, and is usually of short duration. The patient is apprehensive and is afraid to move, while in occlusion the patient is extremely restless and refuses to remain quiet. The angina

pain responds readily to nitrites and soon passes off. In occlusion, the pain is of unusual intensity, requiring large doses of morphine for relief. In this connection one should remember that many patients with a history of angina for some time finally succumb to an attack of occlusion.

In both mitral stenosis and syphilitic aortitis, severe paroxysms of pain are said to occur, but they are rarely confused with coronary obstruction. Although not ordinarily given as a differential point, one should bear in mind that cerebral hemorrhage is sometimes confused with coronary attacks. The onset of the attack, the absence of characteristic pain, the gradual development of coma, the characteristic neurological findings and the extreme rarity of death within several hours, should tend to differentiate these conditions.

The remarkable resemblance of some cases of coronary occlusion to surgical accidents should not be forgotten. The sudden onset of epigastric pain, nausea, vomiting and occasionally board-like rigidity of the abdomen, with signs of collapse, suggest acute gallbladder disease, perforation of a peptic ulcer, pancreatitis. Examination of the heart may give little information. A history of previous attacks of shortness of breath is very suggestive. The presence of rales at the bases offers the chief differential point, as evidence of congestive failure is practically always present in an attack of occlusion of any severity. Levine saw a case operated upon for gallstones which showed coronary thrombosis at autopsy. Herrick, on the other hand, saw a case, operated upon for gallstones, showing coronary thrombosis as well as gallbladder pathology, on post mortem examination.

PROGNOSIS

It is extremely difficult to give the prognosis for the acute attack. Certainly, if the patient survive the first few days, especially if the temperature and white count return to normal, if the signs of congestive failure are lessened, if the heart sounds are of better quality and if there is a gradual improvement in the blood pressure level, the outlook is hopeful. On the other hand, many patients who seem to be making satisfactory progress die quite suddenly while still at rest.

The general prognosis is somewhat more encouraging. In White's series of sixty-two patients, thirty are alive and have been living for periods varying from three months to ten years. Seventeen of them have lived for more than a year. The average duration of life in those living is twenty-four and one-half months. In analyzing his series, White came to the conclusion that "the sex and age at which the attack occurs seems to make little or no difference as far as prognosis is concerned. Hypertension, evident sclerosis and syphilis alter the prognosis hardly at all. Poor heart sounds and congestive failure add to the gravity of the prognosis. . . Neither the previous occurrence of angina pectoris nor its duration prior to the attack of coronary thrombosis has seemed to matter."

The general immediate mortality, according to Levine, was fifty-three per cent. "One might fairly consider that a patient has an even chance to survive an attack of coronary thrombosis." In Levine's series of 143 cases, the average duration of life was twenty-four months, which coincides exactly with White's observations. Levine predicts that the average length of life in his series will probably be three years, with extreme variations of from a few months to ten or more years.

TREATMENT

By far the most useful drug in the treatment of coronary occlusion is morphine. It should be given in large doses to control the severe pain. One-half a grain hypodermically, repeated within an hour, is sometimes required. Most authorities agree that digitalis should be started soon after the attack. It should be given in massive doses so that the patient receives the equivalent of one and one-half grains of the powdered leaf per ten pounds of body weight within twenty-four hours. Nitroglycerine is probably contraindicated. Caffein is useful, especially if Cheyne-Stokes respiration is present. Levine has used quinidine with life-saving results in cases with ventricular tachycardia.

The subsequent treatment is even more important than that of the attack itself. The lives of quite a number of these patients can be prolonged for weeks and even months by prolonged intensive care following the acute attack. The patient should be kept absolutely flat in bed for a period varying from six weeks to six months. The value of prolonged rest cannot be over-emphasized and one month needlessly spent in bed will do no great harm to any patient with coronary thrombosis.

In every case of sudden, severe, precordial or upper abdominal pain the possibility of coronary occlusion should not be overlooked. As Herrick says, "If the possibility of this accident is thought of . . . . . the newspapers will have fewer reports of prominent men prostrated or succumbing to 'acute indigestion' or 'ptomaine poisoning.' Warfield reminds us that "coronary thrombosis is the

one primary cause of sudden dramatic death in an apparently healthy individual."

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MYOCARDIAL INSUFFICIENCY DIAGNOSIS AND SUGGESTIONS OF TREATMENT

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Published statistics, which are supported by prevailing opinion, indicate that there has been a marked increase in both the incidence and death rate in heart diseases during the past twenty-five or thirty years. However, this increase may not be so great as statistics would lead us to believe. There are a number of factors which contribute to this apparent increase: (1) The improved methods of diagnosis, making possible the enumeratian of a larger percentage of diseased hearts.[1] (2) The increase in the span of life, resulting largely from the decrease in death rate among children, thus permitting a larger percentage of individuals to reach the "heart age." (3) Actual increase in heart disease.

"The heart is the body's engine—the most vital and important motor in the world—yet the only one whose requirements and age defects are neglected until cessation of function is not only foreshadowed but threatened or actually at hand."[2] Physiologically the heart is a perfect machine, a complete power unit within itself, with property of independent function, if necessary. The muscle possesses certain inherent qualities which are essential to normal functioning of the organ. These are: contractility, irritability, conductivity, rhythmicity, and tonicity. Disturbance with any of these qualities means an abnormal heart function and, if this disturbance continues for any great length of time, may result in serious disability and eventually in death of the individual. In addition to these specialized muscular qualities, there exists a special conduction system, composed of the sino-auricular node, the auricular-ventricular node, the bundle of His with its two main divisions, right and left branches which divide into ramifications leading to Purkinje's fibers and finally to muscle cells. Under normal conditions, impulses originating in the sino-auricular node are sent out at regular intervals. These impulses cause contraction of the auricles, which is followed by contraction of ventricles in regular sequence. By this action the blood is forced through the body, supplying all the tissue. The heart[3] beating at the rate of fifty to seventy-eight per minute, with the individual observing usual eight hour rest and performing ordinary labor, occupies approximately one-third of time in action (systole) and two-thirds of time at rest (diastole). Physical exertion, infectious conditions, toxemias, emotional disturbances, toxic agents of various types, and other conditions, influence the heart rate. When the rate increases, it does so at the expense of diastole, thus reducing the rest period. In attempting to protect damaged hearts against definite failure, one of the prime objects should be to reduce "time in action," by so doing, increase the resting period.

From an etiological standpoint,[4] diseases of the heart are classified as follows: (1) Etiology unknown. Under this class, I venture to place a group of myocardial conditions which are manifested principally by subjective symptoms. The symptomatology may or may not be supported by physical signs or electrocardiographic records indicating a myocardial deficiency or even a low reserve. (2) Rheumatic heart disease, active or inactive. This group includes these lesions resulting from acute rheumatic fever, polyarthritis, chorea, growing pains, tonsillitis, pharyngitis, purpura, and other infections of this nature. Dr. Paul White[5] of Boston and Douchette Jones of University of Virginia, in a study of three thousand cases of heart disease in New England, found that rheumatic cases comprised 74.4 per cent of all cases under forty years of age and 27.9 per cent of the entire series. (3) Syphilis possibly accounts for two per cent to three per cent of all cases. (4) Bacterial infections—the subacute bacterial endocarditis, most commonly caused by streptococcus viridans. Some of the chronic endocardial lesions may also be placed under this classification. (5) Thyroid—either hyper or hypothyroid state. (6) Toxic—bacterial, mineral and vegetable. (7) Neurosis—a diagnosis which should be used with care and only after a most thorough search fails to find any other factor

which may account for the symptomatology. Like neurasthenia, it is often a screen shielding ignorance. (8) General systemic disease —such as arterio-sclerosis, chronic nephritis, diabetes, emphysema, hypertension, anemia, and so forth. In this group, probably, should be placed those myocardial weaknesses and disturbances resulting from, or in association with, tuberculous infections, bronchial asthma, pulmonary emphysema, and so forth. (9) Traumatic causes—injuries resulting in wide distribution of scar tissue, causing a narrowing of the lumen of the arteries which interferes with the venous return. A case of this type was recently seen at this hospital. The injury, a shrapnel wound right side of the body, had left extensive scar tissue in the right thorax, right arm and right leg. There occurred partial obstruction of right subclavian artery. A diagnosis was made of aneurysm subclavian, due primarily to the obstruction. The heart in this case showed considerable enlargement and other symptoms of increased activity. (10) Congenital development defect. Myocardial disease, coronary lesions, or long continued overloading of the heart resulting from, or in association with, any of the above conditions, in all probability, will eventually result in myocardial insufficiency terminating in dilatation, general edema, and death.

Tradition and habit have led us to think of heart disease in terms of valvular lesions, manifested by usual terminal symptoms. We have been thinking in terms of anatomical changes rather than of functional changes. MacKenzie defines heart failure as a condition in which the heart is unable to maintain efficient circulation during the effort necessary for daily life of the individual. Such condition may be present in the absence of any signs of cardiac enlargement, so far as our present conception goes of normal heart size, with no abnormal heart sounds, and possibly, a negative electrocardiogram. In some of these cases we must rely upon history and subjective symptoms for diagnosis. Care should be observed at all times in order that we may avoid mistakes, especially those that will make invalids of our patients. However, a diagnosis of myocardial insufficiency, followed by a period of watchful waiting for final confirmation, is much safer for the patient than to disregard the symptoms and pass the case up as one of neurosis, later to find that we have a permanently injured heart, which might have been prevented by proper treatment. "Early diagnosis of heart failure and competent conception of myocardial reserve, its peculiarities and therapeutic possibilities are the problems with which we should be concerned in every cardiac patient."

Dr. Lyman Green[2] says that "tonus deficiency is the chief basic source of heart symptoms, subjective and objective, aside from murmurs of endocardial and pericardial lesions. That tonus deficiencies and even the most urgent symptoms of heart failure may arise in absence of any readily demonstrable gross change in heart outline." The conditions affecting tonicity are: (1) Circulating toxins, as in acute and chronic infectious diseases, chronic nephritis, hidden foci of infection, acidosis or the ingestion of certain poisons, organic or inorganic. (2) States of profound anemia. (3) Other conditions associated with decided nutritional disturbances, especially in the visceroptotic group. (4) Degenerative and inflammatory disease of the heart muscle. (5) Impairment of the intrinsic circulation of the heart. (6) Myocardial overstrain, isolated, continuous or intermittent, in the presence of any of the factors elsewhere stated. (7) Emotional crisis. (8) In weakened or unstable hearts, excessive heat or this combined with vitiated air, hot baths or prolonged sweating. (9) Unduly long confinement in bed. (10) Surgical shock. (11) Overdose of digitalis.

A chronic pulmonary tuberculosis, while not specifically mentioned as a factor by any authors referred to in this article, is, we believe, a frequent cause of impaired myocardial tonicity. It appears reasonable to us that that absorption of toxins over a long period of time, associated with a period of inactivity and marked nutritional disturbance, provides an excellent opportunity for development of myocardial insufficiency. Our study of heart function of patients affected with pulmonary tuberculosis, by electrocardiographic study and other means, tends to substantiate such hypothesis. We believe that, prior to the administration of artificial pneumothorax, surgical collapse, or any other procedure which will tend to limit the breathing space of a tuberculous patient, a more thorough study of his heart function should be made. Such a study is also important before prescribing exercise for an advanced tuberculosis patient who has been physically inactive for a long period of time.

The diagnosis and evaluation of myocardial insufficiency, regardless of the cause, requires a complete study of the patient, including examination of lungs, pathology either active or fibrotic, gastro-intestinal function, circulatory disturbance in the extremities, study of the reflex nervous system, a laboratory study of urine, blood chemistry, basal metabolism, vital capacity, and

so forth. This examination may reveal evidence of dysfunction of other vital organs which was previously thought to be due to heart dysfunction. Likewise, the finding of slight disturbance in blood chemistry, laboratory findings of albumin, possibly some casts in urine, should not distract attention from the heart. Such conditions are often a result of passive congestion, due to disturbance in circulation.

The early recognition of any type of heart disease is important, because it is apparent that it is in the early stage that we are able to institute the most effective treatment procedures. There is a tendency among medical men to fear facing the truth, even when cardiac dysfunction is recognized. They justify themselves by saying that we are likely to make neurotics of our patients by calling attention to a weakened cardiac function. We face the same type of argument in the fight for early diagnosis and treatment of pulmonary tuberculosis. The family physician, in many instances, insists on telling the patient that he has suffered a "nervous breakdown," has a "slight bronchitis," or gives some other simple excuse for the symptoms. This mistaken diagnosis, when finally recognized by the patient, causes him to lose confidence in the family physician. The result of an attitude of this kind on the part of the medical profession, can do only harm. The truth, if completely told, about any heart condition, as well as other chronic diseases in their early stages, should improve the mental attitude rather than produce a depressing effect.

The proper diagnosis will include the cause and whether active or inactive, extent of involvement in rhythm and in heart function; also, whether there is impairment of function in other organs which may embarrass the heart or produce symptoms which affect it indirectly.

The fully compensated heart, whether due to endocardial, pericardial or myocardial lesions, should produce no subjective symptoms and show no physical signs suggestive of failure. The normal heart possesses an unusually large reserve power, thirteen-fold, and is, therefore, capable of performing normal function in spite of certain anatomical abnormalities. When an abnormal condition is recognized, it becomes the duty of the physician to determine, as far as possible, the functioning capacity of the heart, in order that he may be properly prepared to advise his patient as to habits, physical activity, and so forth. The stethoscope, although an aid in diagnosis, may prove a detriment in examination of the heart, unless we recognize the fact that heart murmurs do not necessarily mean endocardial lesion, and, when they are the result of such condition, the patient may still live a normal, useful life without even limitations of activity. May we always think and act in terms of cardiac function with due regard for the welfare of our patient.

Dyspnea, fatigueability, digestive disturbance, pain, palpitation, cough, disturbed sleep and vertigo, are the most common subjective symptoms presented in myocardial insufficiency. Each symptom must be considered in detail, as it may be caused by some other disease. Dyspnea, shortness of breath, on exertion, is probably the most common symptom of the group. It may not be predominant; its presence and severity depend largely upon the activity of the patient. It is due to interference with normal exchange of gases in the lungs and is absent when the heart is fully compensated, not present in valvular disease unless decompensation results. Severe dyspnea, especially when associated with cough and wheezing sounds in lungs, is often diagnosed as bronchial asthma. Differential diagnosis is, in many cases, difficult and can be made only by the most careful and painstaking examination. Fatigueability, a common complaint with many abnormal states, when associated with other subjective symptoms, should always lead us to think of heart disease as a possible cause. Digestive disturbance is a still more common symptom, yet one that should direct our minds to the heart. Beware of the epigastric pain and the complaint of "gas" on the stomach, not relieved by belching. The sense of discomfort following a hearty meal will often yield to digitalis and body rest. Pain associated with heart disease may vary from a mere heart consciousness, a sense of heaviness in the precordial region, to the vice-like griping pain of coronary thrombosis. It is not an uncommon symptom with the tired heart as well as with the more advanced degree of insufficiency. Palpitation may mean anything from a "heart consciousness" to auricular fibrillation or paroxysmal tachycardia. A rapid, pounding heart following slight exertion, unaccounted for by infectious diseases or emotional state, is significant and should be carefully considered in connection with the study of myocardial disease. Cough, especially in the elderly patient, is always suggestive of heart weakness and may frequently be relieved by digitalis. Disturbance in sleep, bad dreams, early morning wakening, are not uncommon symptoms of heart disease, especially when the individual is under nervous tension or too

great physical exertion. Vertigo, although a common symptom of other disturbances, is not an unusual complaint with the cardiopath.

Objective symptoms such as cyanosis, edema, enlarged, tender liver, diffuse apex beat, faint heart sounds, variations in blood pressure, rales at base of lungs, are variable findings, depending upon the degree of decompensation present. The blood pressure may be increased, normal or low. A low pulse pressure, in absence of mitral stenosis, is significant. Cyanosis, although common in decompensation, may be caused by exposure to cold, pulmonary infection, and so forth. The slightly enlarged, tender liver is always suggestive.

The x-ray and fluroscope are valuable aids in diagnosis of myocardial conditions. The custom of determining all heart dimensions by location of the P.M.I. or by percussion, is unreliable. The shape of the thorax, the thickness of the walls, the variation of size and position of normal hearts, make it impossible by these methods to determine with any degree of accuracy whether enlargement has taken place or not. The teleoroentgenogram taken at two meter distance at end of inspiration is probably the most reliable method yet employed for measuring the diameter of the heart. The fluoroscope gives formation relative to dilatation of aorta, size of heart chambers, disturbance in tonicity, and other information wihch cannot be determined by the x-ray picture. Many clinicians rely upon the fluoroscope almost exclusively, utilizing the orthediogram as a means of recording heart dimensions and as a permanent record.

In interpreting the teleoroentgenogram[2], the determination of the ratio of the diameter of the heart to the internal chest diameter at the end of inspiration is of importance. Dr. Greene states that a ratio of above forty per cent in the sthenic individual, and above thirty-three or thirty-four per cent in the asthenic, is fairly good evidence that we are dealing with an enlarged heart.

"The electrocardiogram gives the best information concerning the ventricular muscle obtainable: however, a nominal amount of disturbance may exist without change in the record."[3] A cardiac muscle that is below par because of fatigue or failing circulation, will show a changed electrical production and conduction, which will be manifested by abnormal deflections on the cardiogram. A notched P, a widening of Q.R.S. complex, with or without notching, a slurring of the R, a low T wave, are some of the abnormalities noted.

Confronted with the fatigued heart, with its evidence of diminished reserve, or the more advanced type, with its moderate or severe manifestations of insufficiency, our problem is very much the same. We have much to learn about the management of myocardial insufficiency but our course, so far as making an effort to diminish the load on the heart and support the muscular effort, seems evident. We cannot hope to repair a diseased muscle or replace a defective valve, but we can do much toward improving function, provided the heart is not too badly damaged. Restoration of impaired reserve may require only well-directed advice, outlining a course of action which will reduce the physical strain. If there be evidence of infective foci or other sources of toxemia, they should be removed. This may mean the removal of tonsils, extirpation of a gallbladder or thyroid gland, extraction of infected teeth, treatment of pulmonary infection, treatment of diabetes, nephritis and so forth, or probably the study of the colon and elimination of toxic products from this source. Our effort to reduce the demands are especially important when the effect of weakened circulation is shown by secondary renal changes and edema. A low protein, decrease or elimination of salt, decrease of fluid intake, are all factors which will tend to diminish the work of the heart and especially the kidneys.

Decreased physical activity for the patient, with minimal impairment of reserve, increased periods of rest, or, in severe cases, absolute rest followed by a gradual resumption of physical activity, is the most important and effective part of the treatment procedure. Body rest is nature's method for improving muscle tone, and for restoration of myocardial reserve. This remedy, like others, should not be prescribed in too large doses. It is extremely important that we guard against making an invalid of our patient. Rest maintained for too long a period may result in loss of skeletal muscle power, as well as further loss of cardiac power. Rest, therefore, should be as carefully prescribed and resumption of exercise as closely guarded, as is any other part of the prescription. Diet is also an important feature in the treatment program. There are two factors to be considered in prescribing a diet. We desire to limit certain articles of food because in so doing we reduce the work of the kidneys and indirectly the work of the heart; still, the diet must be nutritious, giving ample nourishment to the already crippled myocardium. A simple prescription of low protein, diminished fluid, and low salt is not adequate. We should give attention to the caloric value and

endeavor to supply nourishment which will give the greatest amount of support to the heart with least possible strain on the kidneys.

The utilization of a high carbohydrate diet was probably first advocated by Goustin[6], 1911. Intravenous injections of glucose, combined with insulin, have been used with considerable success, especially in those hearts badly damaged, where patients' intake by mouth is limited. Dr. Albert S. Hyman of New York and Dr. Maurice Protas of Washington, D. C., have recently outlined a series of diets relatively low in protein and fat and high in carbohydrate, which seem to meet the caloric requirement. We feel that these physicians have made a valuable contribution to our treatment procedure. These diets provide the patient with a nutritious, palatable diet and one that is fairly easily handled by the gastro-intestinal tract.

Digitalis, when properly administered, is a very potent remedy in certain types of heart disease. It is frequently employed in an indiscriminate, empirical fashion; many times, no doubt, to the detriment of the patient. It is not indicated as a therapeutic agent in all abnormal functions of the heart. It has certain very definite indications[1]; namely, congestive heart failure as manifested by dyspnea, cyanosis, edema, alone or in combination, which are obviously the result of decompensation; auricular fibrillation or flutter with the ventricular rate high, and as a therapeutic test. Many clinicians have argued that digitalis has no place in the treatment of congestive heart failure (myocardial insufficiency) with normal rhythm. However, the clinical results obtained with digitalis therapy in these cases, while not so outstanding as in the cases of auricular fibrillation, are attested to by White[8], Lutem[9], Fahr[10] Christian[11], and many others. Its beneficial effect in this type of myocardial weakness[12] is, no duobt, due to its specific effect of increasing the amplitude of ventricular contraction by its direct action on heart muscle. In extreme cases there occurs a more or less direct effect on the vagus center, causing a prolongation of the auriculo-ventricular conduction time. In auricular fibrillation and flutter, a slowing occurs, partly from stimulation of vagus center, and partly from depressing action on auricular ventricular conduction system. The direct effect on muscle cells, the improvement of muscle tone and contractility, this improving the circulation, is indirectly the cause of the reduction in rate in myocardial insufficiency. This theory is substantiated by the fact that the rate of non-decompensating heart with normal rhythm is not affected or, if so, only to slight degree.

Digitalis does not restore normal rhythm in fibrillation. In early cases, or possibly those of paroxysmal type, normal rhythm may be re-established under influence of digitalis, but such an effect is probably not due to the action of the remedy but is a mere coincident. It is in these failing hearts, accompanied by disturbed rhythm or greatly increased rate that we see such spectacular results from saturation with digitalis. Maintenance doses must be continued after digitalization is produced, in order to maintain function and a fairly comfortable state.

There are times when we are in doubt as to whether digitalis should be given. In cases with subjective symptoms, especially in elderly persons, or in some cases with heart pains which cannot be accounted for except from myocardial fatigue or beginning failure, we are justified in administering digitalis as a therapeutic test. The action should be carefully watched and the administration stopped as soon as it is determined that untoward symptoms develop or that no benefit is to be derived.

Digitalis therapy is not indicated in compensating hearts except in fibrillation and flutter; in tachycardia due to toxemia; in infectious fevers, such as pneumonia, rheumatic fever, endocarditis or other conditions causing rapid heart action, unless there is a decompensation. It is not indicated in vasomotor collapse such as is frequently encountered in surgical shock. Its use is contraindicated in high-grade heart block accompanied by Adams Stokes' syndrome or in the irritable heart of the so-called "effort syndrome." Hypertensions, premature contractions, hypertrophy, pulsus alternans, are not contra-indications to employment of digitalis, provided there is evidence of decompensation present.

Administration: Freshly prepared, standardized tincture and powdered leaves are available and may be obtained from almost any of the leading pharmaceutical houses. Either the tincture or dried leaf is satisfactory. There is apparently no advantage obtained in prescribing the usual proprietary preparations. Increased potency over the standardized tincture and leaves is usually the claim of the distributors of these proprietary preparations. I believe that the majority of our cardiologists agree with me that no advantage may be expected.

When digitalis is therapeutically indicated, it is usually indicated to point of saturation or digitlaization unless symptoms develop which are a contraindication. The term sat-

uration or digitalization does not imply toxicity, which will be manifested by anexoria, nausea and vomiting, headache, disturbed vision, mental disturbance. Any one of these symptoms should put us on guard, unless they may be otherwise accounted for. The cardiogram is the surest and safest method of maintaining check on digitalis therapy. The occurrence of paroxysmal tachycardia, delayed P. R. conduction time, heart block, are all conditions which indicate digitalis withdrawal—at least to point of non-toxicity.

After the point of saturation is reached, the problem of maintaining such a state over the desired period of time confronts us. The maintenance dosage varies, depending upon size, age, and general condition of patient. Pardee[5], in study of action and rate of elimination of this drug, determined that 0.1 to 0.2 gm. is eliminated within the twenty-four hour period. This being the case, the average maintenance dose would be .15 gm. leaf, and 1.5 c.c. of tincture. This dosage is estimated for patient weighing 150 pounds. Children eliminate more rapidly in proportion to weight.

Dosage: Eggleston, after study of cat unit method of Hatcher, recommends for quick digitalization, twenty-four to thirty-six hour period for individual weighing 150 pounds, an initial dose of 11 c.c. of tincture followed in six hours by 5 c.c.; in six hours by 3 c.c. and thereafter 2 c.c. each six hours until digitalization has been produced. The slower method, however, requiring three to six days, is safer, and more satisfactory in the majority of cases. Roughly speaking, 2.25 gm. dried leaves—22.5 c.c. of tincture—is required to digitalize the 150 pound individual. If the slower procedure is selected, we must add sufficient quantity to take care of daily elimination.

As a complication, or late manifestation, of myocardial insufficiency, we often find it necessary to treat edema, of varying degrees; this condition is usually associated with symptoms of hypostasis in lungs, liver and kidneys, as well as gastro-intestinal disturbance, pulmonary edema, and so forth.

In cardiac dropsy, digitalis may be all that is required to bring about satisfactory renal elimination and disappearance of edema. However, many cases do not respond to digitalis alone, and we are forced to seek more powerful diuretics and more careful supervision of diet and fluid intake is indicated. Some of the medicinal preparations which may be used to advantage are: urea, a safe diuretic to be used in edema in absence of uremia; ammonium chloride, alone or in association with novasurol or merbaphen. The latter preparations are administered in 1 c.c. doses intravenously, first day, gradually increased each second day until satisfactory elimination is obtained or contraindications become manifest.

The remedies mentioned above are all valuable aids; however, we must not overlook the value of complete body rest in the treatment of cardiac failure. If rest cannot be obtained otherwise, then morphine, which is considered one of the best heart tonics, should be employed.

SUMMARY

In this discussion, an effort has been made to stress the importance of early diagnosis of all heart disturbances and to initiate the proper treatment procedure; again to call attention to the fact that murmurs are not necessarily an indication for invaliding the patient, or for instituting treatment; that our action in all cases should be guided by the functioning capacity of the heart; that digitalis preparations are now well standardized —therefore, it is not necessary to seek special preparations in order to obtain maximum benefit. Digitalis, in spite of the more modern teaching of its specific action and therapeutic indication, is still prescribed in an empirical fashion. In treating myocardial insufficiency, one of the most important procedures is body rest, and morphine, used temporarily, is one of the best known heart tonics; also, in treating edema, there are a number of diuretics which may be employed to the advantage of the patient.

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EARLY CANCER RECOGNITION WITH SPECIAL EMPHASIS ON THE CERVIX

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Here, in the semi-arid regions of the Southwest, where congregate the tuberculous from many states, we are inclined to consider tuberculosis the greatest medical problem of the age; and from an economic point of view it still holds first place, attacking, as it does, the younger generation, whose earning capacity is thus greatly impaired. Through a campaign of education during the past twenty-five years, the mortality from tuberculosis has been reduced twenty per cent, and during that same time the mortality from cancer has increased twenty per cent.

According to the Bureau of Vital Statistics of the United States Government, in 1900 cancer was seventh as a cause of death. It has gradually but steadily advanced until today it is fourth as the cause of death, being preceded only by pneumonia.

It is in the hope of checking the ravages of cancer that a wide-spread campaign of education along the lines of early detection and treatment, is being carried out. I believe in the next ten years the profession and public will be as well informed on the subject of cancer as they are on tuberculosis today, and it is up us to enlist the full cooperation of the profession and public in this very laudable endeavor. When that time comes, cancer and certain death will not have the same meaning as it has to many people today. The scientific world knows it, but the truth has not yet reached as many men and women as need to have the knowledge. Cancer does not mean certain death—even though over 110,000 persons succumb to it in this country annually. One of the distressing facts as revealed by statistics is that the more highly civilized and congested the population becomes the higher the death rate. Improved sanitation is not capable of producing any effect and personal hygiene has but a limited application; rich and poor are alike affected. It is, therefore, necessary to attack this scourge in some other way.

The aboriginal people have it all over us on the cancer mortality. The statement has even been made that no full-blooded Indian woman of either North or South America has ever had a carcinoma of the breast, yet over 10,000 women die with cancer of the breast every year in the United States. If anyone has ever seen a case of carcinoma in the breast of an Indian women it would be of unusual interest to report it.

Just as, in the fight on tuberculosis, the family physician and the public had to be instructed to think of tuberculosis in the early stage, so must they be instructed to think and act early in cancer. It would matter little how good your tuberculosis sanatorium if patients were permitted to develop cavities before they were referred for treatment. The same is true in cancer, failure to recognize the condition and act promptly is responsible for a large majority of cancer deaths. It matters not to whom a cancer patient may go for treatment, if the growth has already spread beyond all possible chance of its destruction or removal, it is sure to prove fatal.

The success of the campaign to reach cancer cases early must, as it does in tuberculosis, rest on the shoulders of the family physician. It is he who is the key man in the cancer team. The family physician will be the first person consulted and to him, after the special treatment, the patient will return. The condition and length of life of the returned patient will be determined very largely by the stage the cancer has reached when first diagnosed. This brings us to the necessity for the periodic examination by the famly physician, and in all patients past thirty-five years of age, early cancer is to be kept constantly in mind. Examination on the birthday constitutes an ideal arrangement—let the examination be thorough and complete and a careful record kept of it.

Last spring, while I was acting as a member of the Cancer Committee, in conjunction with the President of the State Medical Association and our County Health Officer, we jointly made talks before Civic and Women's Clubs and at the school houses up and down the valley. We stressed the value of yearly examinations by the family physicians. From the way the talks were received, I feel certain that the public are eager to improve their health conditions. I was told that, subsequently, many people presented themselves for examination.

I wish to cite an example of how not to make an examination: A gentleman, feeling there was something worth while to our plea for periodic examinations, called on his physician and, instead of making a careful examination, the Doctor swapped a few good stories, asked a few questions and then told him he was all right, to go on and forget it. This gentleman went to another physician the next day, told of his experience, was examined and gladly paid for the examination. The physician who slighted his work is one

of those who are hollering their heads off to the effect that the men doing special work are leaving nothing for the family physician to do— while they are neglecting their duty to their patients and the public. It is time that an earnest campaign of preventive medicine be begun within our profession and carried to all branches of society. When the yearly examination has been sold to the public as an economic and life extension measure, they will gladly report for examination. That will mean 119,000,000 examinations — more than three a day for each practicing physician. Then there is the immunization from diphtheria, typhoid and scarlet fever. The field of medicine is far from being well cultivated and, for the benefit of the doctor as well as the public, it is time that we devote a few hours to the acquisition of a little knowledge about our wonderful animal bodies, and how to take care of them. Up to the present, our principal education has come from the insurance companies and, while they have laid the greatest stress on the value of a policy to protect our heirs, they have nevertheless studied their mortality statistics and realize that in periodic physical examinations they have an asset: hence, the Life Extension Institute, and similar life insurance movements.

If I were in general practice today, I would keep a list of the birthdays of my patients and one week in advance of the birthday I would write each one a letter telling him of the value and advisability of a yearly examination. I am sure that it would meet with general approval. The automobile dealer from whom I bought my car sends me a card regularly, stating that the car is due for inspection on a certain date. Most people, I believe, would appreciate receiving as much consideration as an automobile.

At the time of the yearly examination, the family physician, as the key man in the cancer team, will not only detect early cancers but he will do even better, he will discover and remove the conditions which so commonly lead to cancer. Many cancers give us warning a year in advance of the time the cells take on the rapid uncontrollable multiplication without regard to function, that we know as cancer.

Most cancers are preceded by various forms of chronic irritation and it should be the duty of the family physician to set in motion the corrective measures for these conditions when they are discovered, and let it matter not in what part of the body they may be found. Remember, there is no more distressing death than cancer of the rectum, and it is appalling how many are treated for hemorrhoids in their incipiency.

Much that I have said so far can, with equal propriety, be applied to the early development of cancer on the skin, in the mouth, breast, and elsewhere; but what I desire, at this time, is to focus our attention on cancer of the uterine cervix, variously estimated to cause from thirty to thirty-five per cent of the cancer deaths in the female. Undoubtedly, a great many of these patients could be saved by routine physical examination and prompt application of treatment directed to the abnormal conditions frequently present on the cervix as a result of lacerations or infections.

There is probably no accessible lesion, as amenable to treatment, that is so badly neglected, both by physicians and the women themselves. Not all physicians are alive to their duty in these cases. A survey conducted by the Pennsylvania State Cancer Committee in 1910, showed that of the women being treated for cancer of the uterus, ten per cent did not receive a local examination when they first consulted a physician. After an educational campaign of thirteen years, a re-check was made and only seven per cent had at first been treated without being examined. It was found that ten per cent of the physicians first consulted were responsible for fifty-one per cent of the delay in starting treatment. The average delay being nine and a half months. Certainly, as Dr. Pancoast states, these ten per cent have a great deal to answer for. Fifty-seven per cent of the physicians who encountered carcinoma of the cervix permitted of no delay in attacking the growth; many others wasted months in local treatments, thinking they were dealing with benign conditions.

Definite diagnosis of very early carcinoma of the cervix is not easy and it is no reflection on the family doctor when he cannot himself make a positive diagnosis. There is an old saying, and a true one, that the more simple the diagnosis, the more fatal the case. In the very early states, the diagnosis is dependent on a microscopical examination and, while there is an element of danger in removing a section, it is not to be compared to the dangers attendant upon delaying an accurate diagnosis. Unfortunately, very few cases are detected in the early stages. Very early cancer gives no detectable symptoms and can be recognized only by a physical examination. One of the greatest drawbacks to early recognition is a very pernicious belief among women that, during change of life, abnormal discharges and bleeding are to be expected; until this age-old belief has been uprooted, the mortality is certain to be high. Carcinoma of the

cervix begins either as an indurated nodule beneath the surface and without loss of tissue, or as a circumscribed indurated ulcer. The first form must be differentiated from a cyst, scar tissue and myoma. If a cyst is suspected, usually by the bulging and glossing of the mucous membrane, a puncture will reveal clear cystic fluid. Scar tissue will remain constant on subsequent examinations. Scar tissue is always to be regarded as suspicious. Cancer does not develop in scar tissue, but it frequently begins in tissue adjacent to a scar. Small fibroids may feel like a beginning carcinoma, but they are usually multiple and to be found in the body of the uterus. A history of menorrhagia is also important.

An ulcer of the cervix is to be differentiated from erosion due to lacerations with eversion, mechanical pressure either from a pessary or malposition of the uterus, an irritating uterine or vaginal discharge, infection from gonorrhea, tuberculosis, syphilis or chancroid. In each instance, the symptom which brings the patient for treatment will be a vaginal discharge, sometimes streaked with blood. If the ulcer spreads rapidly, it is probably chancroid. A chronic ulcer resisting treatment is probably syphilitic, tuberculous or malignant. Examination of scrapings for spirochetes and tubercle bacilli should establish the absence or presence of the first two, and the time-honored application of a pledget of ten per cent copper sulphate held against the ulcer for one or two minutes every third day, with antiseptic douches in the interval, will usually clear the diagnosis. The bleeding will be increased if you are dealing with a cancer.

The greatest cause of delay in reaching the cancer of the uterus patients early is the failure on the part of the women past thirty-five years of age to realize the serious possibilities of a blood-streaked vaginal discharge the day following coitus, a douche or some unusual effort on the feet; also, their failure to consider a leucorrheal discharge as a disease that should be remedied. So many times the warning signals are ignored because women at the cancer age become accustomed to menstrual irregularities and they have not been instructed as to the seriousness of the "spotting" symptom.

It is my hope that, as a result of the discussion this paper may provoke, we will return to our respective fields of endeavor and do more than we have in the past to educate our clientele to submit to a yearly physical examination, this examination in women to include the pelvic organs, and that, in the event any precancerous condition is found, we will see that the same is promptly removed and a proper hygiene established. Certainly, this is not too much to ask of us who are entrusted with the physical welfare of those who support us. When we stop to think that, of the women who enter our office and who die between the ages of forty-five and sixty-five, one out of five will die of cancer, we cannot ignore the importance of keeping malignancy constantly in mind.

DISCUSSION

DR. R. B. HOMAN, El Paso: I should like to emphasize the remarks about physical examinations. Recent experiences have convinced me that people are anxious to have doctors tell them what to do; they want to be told how to protect themselves from disease and prolong lives. The diseases in which the mortality is being reduced are the infectious diseases, where the results depend on the work of the physicians. Those diseases which depend on the education of the people are the diseases which are increasing in mortality.

DR. E. PAYNE PALMER, Phoenix: Dr. Cathcart's paper covers a very important subject, the importance of which should be fully realized by the medical profession and the laity. With a death rate, from cancer, of 100,000 in the United States annually, it is time to sit up and take notice. Unfortunately, the onset of cancer is insidious and frequently symptomless for a long period of time. If it were not for this fact, the urgency of emergency treatment for all cases of cancer might be realized both by the medical profession and the laity.

The "pathological cervix" is frequently the source of metastatic infection and is usually overlooked, while teeth and tonsils are removed and the sinuses treated for infection. The pathological cervix" which is due to neglected lacerations at childbirth, can, and should, be prevented by immediate repair of the laceration. When done in a hospital under strict or aseptic precautions, satisfactory healing will follow and a pathological process will be prevented.

Nonspecific infections of the cervix and cervical mucous membrane are, in the majority of cases, due to lacerations that result in erosions, ectropion, cysts of the Nabothian gland and hypertrophy of the lips. Ulceration of the cervix must always be regarded seriously and if it does not heal promptly after appropriate treatment, biopsy should be made. Chronic cervicitis of the ulcerative type, with cellular changes and rich in mitroses, constitute a precancerous condition and favorable environment for the development of malignancy. Erosions, unhealed lacerations of the cervix and a cervix that bleeds easily should be considered precancerous.

Cancer of the cervix occurs almost exclusively in cervices that have had some inflammation or traumatic lesion, usually the result of childbirth. It is estimated that about ninety-eight per cent of women with cancer of the cervix have had children.

A very careful physical examination is not sufficient to make the diagnosis in any of the very early cases of cancer of the cervix. Biopsy should be resorted to more frequently, as there is no other way to make diagnosis in the very early stages of cancer of the cervix. As soon as the diagnosis of cancer is made, the case calls for emergency treatment. Watchful waiting means certain death.

DR. G. WERLEY, El Paso: I am strongly in favor of everything advocated in this paper and discussion. I do not feel that you can scare people too

much. Dr. Cathcart's paper is of extreme value in pointing out the fact that cancer, or diseases leading to cancer, taken early are curable. Since the discussion has broadened into the general one of education of the public, I am led to inquire in what manner the public can be educated in heart disease. The first thing is to educate the doctor; get the doctor to study heart disease so that he will be prepared to give advice on the points raised. Until you get the doctors prepared, you dare not tell the people to go to the family doctor, because they will not get the proper advice.

DR. WILLIAM DOCK, San Francisco: It seems to me that, in preference to the periodic health examination, it is much more important to educate people to come for examination regarding the first symptom which indicates illness. This is better than a superficial examination once a year. It is much easier to do a biopsy on a patient who comes with the first symptom, than it is to do this on a patient coming for examination.

DR. CATHCART (closing): It will be interesting to observe what will be the effect of cigarette smoking by women on the incidence of cancer. Also, to note the effect of the abolishment of the corset on the incidence of breast cancer.

CHRONIC PROSTATITIS

H. M. PURCELL, M. D.
Phoenix, Ariz.

(Read before the Maricopa County Medical Society, at Phoenix, Ariz., at their meeting in December, 1929.)

The title limits this paper to the chronic inflammations of the prostate gland. The chronic hypertrophied prostate is not included in this paper, although by some it is considered an inflammatory condition.

By far the majority of the cases, probably about 95 per cent, are of gonorrhoeal etiology. The next most frequent class are those following a gonorrhoeal infection but in which the gonococcus has disappeared, the inflammation persisting because of secondary infection probably due to improper treatment. The next most frequent class are considered as due to focal infection elsewhere in the body. The other class is tuberculosis of the prostate which happily does not make up a large per cent.

The symptoms complained of consist of the "morning drop," backache and indefinite pains in the perineum and penis and, at times, in the groins and thighs. Occasionally there will be urgency and frequency of urination, rheumatoid or arthritic pains and cloudy urine.

The findings on examination show, in about one-third the cases, some abnormality of the prostate to palpation. It may be enlarged but there is normally considerable variation of the gland, but as a rule the size corresponds to that of the patient—a large man normally having a larger gland. There may be irregularity in contour—one side larger than the other. Nodules may be palpable in the gland and are usually due to chronic infection but may be caused by stone or cancer. Frequently we find a soft, boggy feel to the gland which is very suggestive. Tenderness is present in about half the cases. Here again we must be cautious, as normally many men are very sensitive to rectal examination, especially to pressure on the prostate and may complain of much pain when in reality there is no abnormality demonstrable of an infectious nature. Again there may be a prostate loaded with pus in a non-sensitive type of individual who will evidence no sign of tenderness even on hard pressure. Experience alone will allow one to reach a conclusion.

Examination of the urine shows a clear first glass; a cloudy first and clear second; or all cloudy, depending on the activity of the infection.

The most important evidence of prostatic infection, and the only one on which to base evidence of cure, is the prostatic smear. It is important that this be taken properly and, still more important, properly interpreted. Care must be taken not to strip the seminal vesicles as this would dilute and render examination of the prostatic fluid practically worthless. The seminal vesicles cannot, as a rule, be palpated in the normal state, but inclusion of their secretion in the specimen can be determined in the normal condition by the presence of spermatozoa. At times the seminal vesicles seem to lie so low over the prostate as to make it impossible to get a true prostatic smear. The bladder should be emptied of urine, by voiding before massage, for two reasons; so as not to get urine in the prostatic specimen, and in order not to contaminate it with secretion from the anterior urethra. A little pus in the anterior smear signifies little but the same amount in a prostatic smear may mean a considerable prostatic infection. The prostate is emptied by the downward stroking of the finger in the rectum with the patient bending over at right angles at the hips. The last stroke should be in the midline and continued downward to force the expressed secretion through the cut-off muscle. The glans penis is placed in a medicine glass held by the patient to catch the expressed prostatic secretion. Often the major part of the secretion will accumulate in the bulbous urethra which acts as a sort of pocket but this is easily emptied by gently stroking the urethra from the perineum to the external urinary meatus.

The secretion so obtained is smeared fairly thick on a slide and allowed to dry. Some prefer to examine the specimen in the wet state for pus content. I prefer the stained

specimen, using simple methylene blue stain. Here again difficulty is encountered in obtaining a good stain, for if the secretion is not fixed to the slide by sufficient heating it will wash off when rinsing off the stain, and if heated too much it coagulates so as to be unfit for examination. For this reason, I always prepare two slides of each specimen and in about one-third the cases have to try the second slide to get a good stain.

On microscopic examination under the oil-immersion lens, the presence of more than five leukocytes per field indicates a definite infection, but, as a rule, a normal secretion will not contain more than one leukocyte to several fields. This questionable zone up to the five cell point seldom concerns us, for, as a rule, the leukocytes are much more numerous or the field is normal in leukocyte content. Another thing indicative of a normal secretion is the presence of lecithin granules which we rarely see in the presence of infection. Frequently, on the first examination of a smear from a patient, portions of the smear appear practically normal but we find clumps of cells scattered about; these are usually the very chronic cases and on beginning active treatment the leukocyte content will increase for a time but there will be a more even distribution. The presence of spermatozoa should be noted to determine if specimen, at least partially, came from the seminal vesicles.

The bacterial content of the smear is of much less importance than the leukocyte content. Gonococci are very hard to find in prostatic fluid even in the average case of fairly acute prostatitis. Other bacteria are likewise scarce, but, as a rule, the type of infection can be found if searched for, but we must always bear in mind the danger of a contaminatioin from the urethra, especially the external uninary meatus. In tuberculous infection, the tubercle bacilli can usually be demonstrated, at least if a series of smears are taken. Cultures, as a rule, are worthless except for vaccines as the possibility of contamination is too great.

The diagnosis depends on rectal palpation of the gland, the prostatic smear, the urethral smear, examinatioin of the urine, history and at times cystoscopic examination. Examination begins first with an attempt to strip some secretioin from the urethra for a smear. Examination of this will as rule show the gonococcus if the case is of gonorrheal etiology. It is often well to give the patient a slide with instruction as to obtaining a smear of the "morning drop" which will often show the gonococcus. Other bacteria have little bearing on the etiology of an accompanying prostatitis but a positive gonorrheal finding practically establishes the diagnosis. The patient then passes the urine in several glasses. All the urine may be clear and there still be prostatic infection in a very dormant condition. As the rule, the first glass shows a slight cloud which may come from the anterior or posterior urethra or bladder. With a clear second glass, it comes from the urethra, anterior or posterior, or both. If all glasses are of the same degree of cloudiness, the condition is all prostatic, bladder, or above the bladder. As a rule the first glass shows a denser cloud showing the infection in the urethra also. At times the last glass shows a denser cloud which is due to pus in the base of the bladder or a stripping out of the pus from the prostate as the bladder collapses, the pressure exerted to force urine from the bladder apparently extending to the prostate gland which is closely apposed to the base of the bladder and interwoven with its structure.

The above conclusions apply only in case the cloud is due to pus. On every examination where there is a cloud present, acetic acid should be added to clear up any cloud due to alkali and in this locality about half the cloudy urine is due to alkali, which is not harmful but must not be confused with a cloud due to pus. At times there may be a cloud due to spermatic fluid and at times a bacteruria which must be determined by the microscope, but the latter two are rarely encountered.

Next, the prostate is palpated per rectum and may show any of the abnormal conditions mentioned above, but usually no definite evidence of infection will be perceptible by this method. The prostate is then stripped to obtain material for microscopic examination. The seminal vesicles should also be examined to determine if palpable but are not stripped. Rarely is there an infection of the seminal vesicles accompanying the prostatic infection, at least, that we are able to determine and happily, too, as infection of the seminal vesicles are very refractory to treatment.

Examination of the prostatic secretion microscopically is the most important part of the examinatioin and on the leukocyte content is based the presence or absence of infection. The type of infection is not so easy to determine for, as a rule, the bacteria causing the trouble are not in evidence on the smear and even when found, with the exception of the gonococcus, are not conclusively the cause of the infection; they are so scarce as uusally to suggest a contamination. The urethral smear, if positive for the gonococ-

cus, is practically conclusive as to the nature of the prostatitis.

Cultures, as stated before, are practically worthless; the gonococcus will hardly grow at all and the positive cultures of other bacteria are more often than not contaminations. The history of previous urethral infection may determine the gonorrheal nature, but even here, the gonococcus should be found before beginning active treatment. The complement fixation test is of practically no value and at present I rarely use it. A positive test is very suggestive, but I would not consider it conclusive evidence of the gonorrheal nature of the infection. A negative test would also suggest that the condition is not gonorrheal. However, the results are not conclusive, and, as the treatment varies little in the different types of infection, it is rarely worth the cost to the patient. Furthermore, the previous use of gonorrheal vaccine renders the results more doubtful,

The prostatic infection may be the result of imporer use of syringes or instruments during a case of gonorrhea, resulting in the implantation of secondary infection in the prostate, the gonococcus itself having disappeared. Cases without a history suggestive of a urethral infection are usually ascribed to focal infection as a cause, and, in about half these cases, other focal infection is demonstrable and in perhaps half of these there is improvement of the prostatic condition following its removal.

At times there is probably a localization in the prostate of an acute systemic infection which does not clear up with the systemic condition but remains as a chronic prostatitis. Likewise, constipation is probably a frequent cause of trouble both from absorption of colon bacilli and because of congestion in the prostatic region due to the pressure of the fecal mass.

We also have tuberculosis of the prostate which, as a rule, is secondary to tuberculosis either in the epididymis or the kidney. Hugh Young claims the primary infection to be in the prostate, but he is practically alone in this contention.

Treatment depends on etiology. I will consider first the gonorrheal type. Drugs by mouth are practically worthless; the only effect obtained with antiseptic drugs is during the passage of the urine through the prostate on urination, which is too fleeting, in the concentration possible, to be of any value. Sedative drugs in conditions that are at all acute are worth while; e. g. hyoscyamus and the alkalies and possibly oil of sandalwood. Pyridium, hexresorcinol and all the other drugs for oral administration are, in my opinion, absolutely worthless; likewise mercurochrome intravenously.

Rest is an important point, physical and sexual; the latter perhaps is a little more important. Alcohol must be prohibited. Moderate physical exercise is permissible but such exercise as riding a motorcycle or truck is very detrimental.

Local treatment is the important method. Prostatic massage is, I believe, very useful but should be done gently and should never at any time cause pain. As the condition improves, massage can be increased, the stimulation to the gland probably being more important than any results from emptying it of its contents. Massage is followed by bladder irrigation, usually of silver nitrate beginning with a quart of 1:10,000 solutioin and increasing the strength of the drug as the reaction of the patient will permit. I believe silver nitrate to be the most important drug we have for local use in genito-urinary infection. It apparently is not only the antiseptic value of the drug, but a peculiar favorable reaction produced on the mucous membrane of the genito-urinary tract, that is efficacious. The following day the patient takes a deep injection to the bladder of mercurochrome which he retains for an hour. If there is any anterior urethral infection, he also takes two or three anterior injections a day which he retains five minutes by clamping the meatus. Begin with one-fourth per cent mercurochrome and run up to one per cent for posterior injections and two per cent for anterior injections. I am particular that the Asepto outfit be used, which insures the use of a sterile syringe for his injection. Other drugs, as acriflavin, potassium permanganate, protargol, etc., are sometimes employed, but, as a rule, main reliance is placed on silver nitrate and mercurochrome. All solutions and apparatus must, of course, be sterile and the silver solutions must be in distilled water.

Vaccines are a routine but their value is doubtful. A stock vaccine is preferred, straight gonococcic for gonorrheal cases and mixed vaccines for other cases and old gonorrheal cases. Begin with small doses (.1c.c.) and increase as permissible without getting more than one inch area of hyperemia, injections every other day. A maximum dose of one cubic centimeter (five billion gonococci) is kept up as long as treatment continues. Autogenous gonorrheal vaccines have not been used, and I have read no favorable reports on them but theoretically they appeal to me.

Surgery has at times to be resorted to for prostatic abscess but that rarely concerns chronic prostatitis. Any strictures present

should be dilated and a prostatic infection will at times not clear up until this is done.

Of physical agents, light and heat have been used. The use of ultra-violet light has been recommended but in my experience has been of absolutely no value. Hot rectal irrigations are of much help in acute prostatic conditions and may be of some benefit in chronic conditions. Diathermy to the prostate is merely another form of heat and although used rather extensively, is of doubtful value. Certainly there is no germicidal effect. Infra-red radiation is another type of heat,—incidently the electric bathroom heater is a dandy,—which is very penetrating and of some slight value but more in acute condtiions.

In the non-gonorrheal prostatitis, the treatment is practically the same except the use of a mixed vaccine to replace the plain gonrorheal vaccine. I have had one case resistant to all other treatment that cleared up nicely on an autogenous vaccine from the prostatic secretion. Focal infection is sought out and eliminated if possible and I have seen definite favorable results thereby.

In the tuberculous prostate, little can be hoped for from local treatment, mercurochrome deep injections seem to give more relief than anything. Removal of the infected epididymis or kidney is indicated if possible. Prostatic infection, otherwise, is practically, if not always, curable, but the treatment often requires one to four months, occasionally longer. During the course of treatment the urine clears first and there is, as a rule, a gradual lessening in the amount of the pus present in the prostatic smear. This at times drops off suddenly to normal.

A normal prostatic smear, with another normal smear in a week, means that the patient is cured of his prostatic infection. I have yet to see a case of gonorrhea which had extended to the prostate, which was not cured of his infection on obtaining normal prostatic findings. The reverse is not the case; frequently have I seen cases with viable gonococci still in the prostate with all urinary and urethral findings negative as to gonorrhea, and unfortunately at least two of these were patients who had been examined before marriage by physicians and pronounced cured, only to transmit their infection to their brides. This could have been prevented had a prostatic smear been taken, as above directed, and every case of gonorrhea should have a negative prostatic smear as regards pus content before being pronounced cured of his infection.

TREATMENT OF GONORRHEAL OPHTHALMIA

HARLEY YANDELL, M. D.
Phoenix, Arizona

(Read before the Maricopa County Medical Society, at Phoenix, Ariz., December 2, 1929.)

Have you ever thought of the number of people in the United States today who are blind, and do you know that nearly twenty per cent are so from some form of gonorrheal infection? I will speak mainly on the treatment of this disease at this time.

Probably the number of different drugs recommended for the treatment of this dread disease would run into the hundreds, and are recommended by as many different physicians throughout the world. Yet, today, we are earnestly and solicitously inquiring about a cure that will cut the infection short and rob the pathological process of its dreadful sequelae,—corneal ulcers with consequent opacities, ruptured globe, staphyloma and final darkness.

Gonorrheal ophthalmia is an acute and severe form of pustular conjunctivitis, caused by the infection from urethral discharges containing the gonococcus of Neisser. Most physicians are familiar with the severity of this dread disease and the long drawn out, drastic and detailed treatment necessary to prevent total blindness, so I will say practically nothing concerning the pathology and the older and universal methods of treatment, but will give in detail the treatment which has proven one hundred per cent effective in the small series of cases I have had.

It is with considerable reluctance that I am reporting this series of cases as I had hoped to try my method on more infections, but since they are considerably scarce in this section of the United States and because of the satisfactory and astounding results in such a serious disease, I felt that I would not be doing my duty to the medical profession should I wait longer. Some of you will perhaps recall my reporting a case as early as 1926 here before this society.

The technic is very simple, yet it must be exact and is as follows:

The eye is cleansed of as much pus as is possible, with the idea that the rays must come in direct contact with the tissue involved. The conjunctival folds are everted (the great swelling of the lids made this very difficult in two cases) and the long pharyngeal quartz applicator on mercury ultraviolet lamp that has been standardized, is held almost touching both the upper and lower lid conjunctiva, averaging a duration of forty seconds each. This is the time required for

my lamp. The patient is directed to look either up or down, in or out, which will avoid the rays coming in contact with the cornea, which should not be treated with the rays unless it is involved. In case of corneal ulcer, which occurred in only one of my cases, I employed the usual treatment, and the ulcer disappeared as rapidly as did the conjunctival infection. This part of the treatment is followed by the usual sovereign remedy, two per cent silver nitrate, with instructions to irrigate the eye every hour with boric acid and instill one or two drops of two per cent mercurochrome solution every two hours. I used the latter part of this treatment years ago, with the patient in bed in the hospital with day and night nurses and the usual time required for cure was three to six weeks.

The ultraviolet ray treatment was given each day in four cases which involved six eyes, and the number of treatments was as follows: One case required four exposures before the pus disappeared, one required five, another three and another only one.

Case 1: Referred by Drs. Bailey and Robb. Male child, three and a half years old, both eyes involved, lids tightly closed due to great swelling, profuse creamy pus running down each cheek. Pus examined and found to be full of gonococci. Cornea not involved. Child seemed to be in great distress, but was far from equalling the apparent distress of the parents. The first treatment was given at 6:30 p.m. Sept. 22, and by three o'clock the next morning the anxious mother, who carefully watched and treated the child each hour, stated that it first began to open the eyes, and when seen at my office about 10 a.m. Sept. 23, both eyes were about three-quarters open and I gave only one more treatment. Eyes were completely cured in six days and with no sequelae.

Case 2: Male adult Mexican, one eye involved. Swelling was tremendous and pus profuse as we usually see in such cases. One treatment was given at the office and the usual home treatment directed. Patient was seen the next day and the swelling and pus were all gone. No further observations were made as he never returned.

Case 3: Male Mexican, age 23, referred by Dr. Garrison. Both eyes involved, right much worse than the left. Patient claimed to be in great pain which was due largely to a right corneal ulcer. This case received five office treatments, in conjunction with the home treatments which extended over a period of eleven days. He claimed he had been treated about ten days before I saw him.

Case 4: Referred by Dr. Charvoz. I think this was the most pitiful case I ever had. A little girl, five years of age. She had a severe vaginal infection and consequent gonorrheal peritonitis. Her abdomen was so distended that she could hardly breathe and each breath, which was so very rapid, was accompanied by an audible grunt. She could not sit up and it was necessary for her to be held in a reclining position by the nurse long enough to take the ultraviolet ray treatment. One eye was involved, and the pus was very profuse and the swelling great. Laboratory findings showed gonococci. Four treatments were given, one each day, and at the end of eight days her eye was completely cured and with no sequelae.

DISCUSSION

DR. H. T. BAILEY: Dr. Yandell has not told half of it. We sent him several patients during 1929. Many times for various reasons we did not want these cases around our office, mostly on account of others fearing they will contract this disease. There is very little danger, however, as some of our best authorities claim that soap will kill all gonococci it comes in contact with. So all that is necessary is to wash the hands thoroughly with soap and water.

There are a number of germicides for gonococci. A temperature of 32°F will kill them almost instantly. A temperature of 107°F will kill them almost instantly. They live and grow in a temperature from 77° to 100° F. and in the presence of moisture. The advocates of physiological saline icepacks can only secure a temperature of 38° F at the most, and that only retards them. Heat of 107° or more in the eye will destroy them.

Silver nitrate will kill them but it leaves a coat of silver albuminate on the lids after applications; too, the liberation of nitric acid in the eye is an irritant. Permanganate of potash is rather uncertain. Sun light will kill them but the quantity or dose is hard to control, and it is very likely to cause a severe conjunctivitis or cellulitis and if it shines directly on the macula it will likely cause blindness.

Ultraviolet light is more nearly a specific and, if handled carefully, is not harmful. These rays are probably nature's greatest sterilizing agents. They are the rays lying between the visible violet and the x-ray zone; they keep air and water pure and doubtless prevent many epidemics. They are the rays between 2900 and 3200 Angstrom units. These are only about two per cent of the rays given off by the sun and vary on account of dust, smoke and other gases in the air, as much one hundred per cent from day to day while they constitute a large percentage of the rays given off from a mercury quartz lamp. They are absorbed readily by oxygen. It is ultraviolet light in rarefied air, that causes mountain air to be more invigorating than air of lower altitudes.

Because of the concentration of ultraviolet rays from a mercury quartz lamp and because of the almost perfect control of the dose or the amount given, this treatment is so effective as a local germicide. It is effective not only in gonorrhea but in tonsillitis, Vincent's angina, sinusitis, nasal and aural polypi and many other surface infections. Some one of the fluorescent dyes, as mercurochrome, acriflavin, gentian violet etc., applied to the surface before the treatment makes the ultraviolet rays more germicidal because the dye seems to carry the ray to the diseased cell.

INTERNATIONAL CLINICS:—Edited by Henry W. Cattell. Volume II, Thirty-Ninth Series, 1929. J. B. Lippincott Company, Philadelphia and London.

This volume contains an unusual number of interesting articles. Perhaps the article which is most striking is "Manners and Morals;" this is a question of medical ethics by Dr. Lewellys F. Barker, a lecture to medical students. The possibilities are that most of us physicians will profit by reading this article.

Other interesting articles in this volume deal with chronic nephritis, the periodical physical examination, inflammatory diseases of the liver, the aged prostate, treatment of pneumonia, diagnostic value of some reflexes, pyelitis, etc.

O. H. B.

DIAGNOSTIC FORUM

CASE OF DROWSINESS AND SWELLING OF THE FACE

(Case 14411, Case Records of the Massachusetts General Hospital, New Eng., Jour. of Med., Nov. 29, 1928. p. 1106).

CASE HISTORY

An Irish-American widow sixty-five years old entered the hospital May 13 complaining of dizziness and slowness.

Four years before admission she had an attack of erysipelas of the face and region of the left ear. Ever since this time her cheeks had lost color and the skin about her eyes had been puffy. She had had three later attacks of erysipelas. For three or four years she had had increasing weakness and slowness in movement. For three years her skin and hair had been growing dry and the circulation in her hands and feet, always poor, had been getting worse. She had been hoarse, although she never had sore throat. In the past two years she had had a slight general loss of hair. For a year she had had a tendency to trip, and had been dizzy after quick motions of the head or when she looked at moving pictures. During the past year her hearing, eyesight and memory had been growing poorer. Recently she had had a tendency to fall asleep, and slept so much during the day that she did not sleep well at night. During the past month her hands had grown very swollen, spade-like and blue. In the past two weeks she had had some dyspnea when she was nervous or hurried. She urinated two or three times at night, and lately had had some urgency. She had lost a few pounds in weight.

Her father died of tuberculosis while she was living with him. One sister had had a nervous breakdown following an unfortunate investment.

The patient had had three or four miscarriages at three months and lost one of her two children the day after birth. She was operated upon for falling of the womb after one delivery. All her teeth had been extracted for pyorrhea. She had some palpitation on exertion or excitement. For years she had had swollen joints in the hands and occasional pains in the hips, due she thought to arthritis. She had gas, relieved by hot drinks. She ate no meat because of high blood pressure. For two years her bowels had required laxatives.

Clinical examination showed an obese woman, asleep and hard to arouse. Voice low, speech slow. Skin cool, subicteric, dry and scaling. Hair coarse and dry. Face rough, cheeks red. Swollen loose skin about the eyes, which opened with difficulty. Whitish secretion on lids. A few keratoses over the body. Superabundance of tissue over the clavicles and upper borders of trapezius muscles. Tongue very large and long, somewhat smooth. Thickened tonsillar pillars and uvula. Small firm axillary glands. Breasts atrophic. Apex impulse of the heart not seen or felt. Left border of dullness 5.5+(?) centimeters, midclavicular line 8.25 centimeters, right border, 4.5, supracardiac dullness 6. (Heart displaced to the right?) Rate regular. Sounds distant. A rough systolic murmur, faint in the apical region, becoming loud in the second right interspace. Left pulse greater than right. Artery walls thickened and tortuous. Blood pressure 195/110 to 145/100. Lung signs: Left chest anteriorly, flatness, diminished or absent tactile fremitus, distant bronchial breathing, egophony, increased whispered voice, over lower portion; no rales; medium crepitant rales over base posteriorly. Abdomen protuberant and tympanitis. Liver edge felt. Rounded mass in region of gall-bladder. Hypertrophic arthritis of phalangeal joints. Pitting edema of ankles and shins. Dorsalis pedis arteries not pulsating; feet cold. Foul leukorrhea. Vagina presented small irregularities. Small non-bleeding hemorrhoids. Pupils and knee-jerks normal, ankle jerks absent. Fundi: disc outlines indistinct but not choked. Vessels not clearly seen. No obvious sclerosis. No hemorrhages.

Urine: amount 28 to 30 ounces on the two occasions recorded, specific gravity 1.016 to 1.026, no albumin. sediment of a catheter specimen negative. Urine culture: colon bacillus. Blood: 8,000 to 6,500 leukocytes. 66 per cent polymorphonuclears, hemoglobin 65 to 70 per cent, reds 3,400.000 to 3,700,000. no marked achromia, moderate anisocytosis, platelets normal. Hinton negative. Icteric index 3. Non-protein nitrogen 25. Stools: guaiac strongly positive at one of two examinations. Basal metabolic rate minus 26, pulse 73, weight 62.9 kilos.

Temperature 96° to 98.9°; usually 98° or below. Pulse and respirations normal.

X-ray examination showed arthritis of the terminal phalangeal joints and the spine, also changes suggestive of gout. There were no visible gallstones. The sella turcica was normal. There was considerable calcification in the region of the pineal gland. The left lung field showed dense dullness except for a small area at the apex. The upper margin of dullness was sharply defined. The outline of the diaphragm and the border of the heart were obliterated. The heart and mediastinal contents were displaced to the right. The right lung field was obscured by the heart shadow, and was less radiant than normal along the borders of the heart and in the region of the base.

The patient was disoriented at night and on the morning of May 18. The edema of the eyelids became marked. The veins of the arms were so engorged that the venous pressure was sufficient to push the piston out when blood was withdrawn. The lips, face, hands and feet were cyanotic. A lumbar puncture was done. The fluid was clear, straw colored, initial pressure 165, jugular compression 610+, release 195, after withdrawal of 5 cubic centimeters 160, after withdrawal of 5 more 140; two red blood cells, one leukocyte; Wassermann and Hinton negative; colloidal gold 0111100000. A chest tap gave 80 cubic centimeters of very slightly opalescent yellowish fluid. On the same day the red blood count was 4,400.000, the blood serum total protein 6.98 grams in 100 cubic centimeters, the hematocrit 38.1 per cent.

That evening the patient seemed brighter and better oriented. The next morning, May 19, she died.

The following discussions of this case were given before the Yavapai County Medical Society and Medical Officers of Fort Whipple, at their regular meeting of Dec. 10, 1929:

DR. J. T. MALONE

We are confronted with several possibilities in the differential diagnosis of this case.

If we consider pulmonary tuberculosis, we have a history of contact and pleural effusion. It is possible, but not very probable, that a well developed woman of sixty-five years would die of tuberculosis. Yet we must consider pulmonary tuberculosis on the basis of that effusion. Considering the history of contact with a tuberculous father; there is disorientation, symptoms suggestive of brain congestion or tumor, (tuberculous meningitis?). However, the fluid was not under pressure and we are told there was no evidence of choked disc; colloidal gold curve and the cell count do not indicate tuber-

culous meningitis. There is a shadow in the left chest, practically a curtain, and we do not know what is going on behind that curtain. They may find at autopsy, as in a previous case, that there is a large cavity behind this dense shadow, which we are interpreting as pleurisy with effusion and a densely thickened pleura. We can not definitely rule out tuberculosis, but we do believe the effusion can be explained in a very logical manner and that tuberculosis, either pulmonary or meningeal, is not a definite factor.

Syphilis: There is a history of several miscarriages and we have suggestive mental symptoms. They tell us the spinal fluid is straw colored. This seems to be a factor to consider later. We believe we can explain the straw colored fluid on another basis. There is a definite negative spinal and blood Wassermann, which is corroborated by the negative colloidal gold and cell count. These apparently rule out syphilis in any form.

Malignancy: We are still more or less speculating. This chest has evidently been a puzzle to the x-ray men themselves. We do not intend to make a diagnosis of malignancy. There is pressure in the region of the aorta, the left pulse is greater than the right. We propose to explain this pressure on a more logical basis than malignancy, because the x-ray findings are so very indefinite. This is an obese woman who gives no history of cachexia; she is subicteric, but not cachectic. There is a tumor in the region of the gall bladder. We would rather interpret this mass as a hydrops of the gallbladder. We are again confronted with tumors, or tumor like structures, in the vagina. We believe the evidence and x-ray findings do not warrant a diagnosis of malignancy with metastases.

There is a suggestion of encephalitis,—drowsiness, thick speech, etc.,—but the leukocyte count is apparently normal. There is no episode of pyrexia.

We believe this a typical picture of myxedema. We would interpret this pleurisy and effusion as myxedematous in character rather than tuberculous.

There is a suggestion of pernicious anemia; there is some distortion of red cells, but the blood count is not typical and the index does not confirm it. They do not tell us that the patient has been under treatment and that this is a remission blood picture. The blood picture would indicate a secondary rather than a primary anemia, with the exception that she may have been under treatment.

Nephritis and uremia: The catheterized urine was negative, no albumin; non-protein nitrogen within normal limits. It is possible there could have been a passive nephritis without albumin and non-protein nitrogen not elevated at that time, but we do not believe we are dealing with that condition.

We are told in the history that there is a soft systolic murmur over the apex transmitted and heard very loudly over the aorta. We read that backwards and it sounded very much better to us. The interne, listening at the apex first, heard the murmur and traced it to the base. We have, then, a systolic murmur loud over the aortic region, heard faintly over the mitral area. We are told by some of the best cardiologists today to dismiss all apical systolic murmurs. We would dismiss this if he had not mentioned that it became louder over the aortic area. The transmission is not to the axilla. It is impossible to make a diagnosis of mitral insufficiency based on a systolic murmur alone, without enlargement of the left ventricle, etc. Heart measurements do not denote enlarged or dilated heart. Percussion measurements and x-ray tell us that the heart is displaced to the right. With such a distortion of the chest as we have here with fluid and possibly air, we can not expect percussion to be of much value.

The systolic murmur over the aortic area suggests aortic stenosis. There is no mention at all of a diastolic murmur. Authorities tell us that aortic stenosis rarely exists alone; if you examine carefully you will pick up a diastolic murmur; where there is stenosis there is insufficiency. Aortic stenosis with aneurysm fits our blood pressure evidence,—the pressure was so high in this case that the plunger was forced out of the vein. Left pulse was greater than right.

If we consider aneurysm associated with aortic stenosis, the picture would seem a little more plausible, but you do not get a pulse pressure such as in this case,—systolic 195, diastolic 110. The pulse pressure rather fits the picture of aortic insufficiency. Either from lack of data or purposely not mentioned, we do not have mention of Corrigan pulse or an electrocardiogram. Aneurysm at the arch, causing pressure on the superior vena cava or innominate vein to account for pressure in the left arm and for venous pressure in general, would still be suggestive of lues. Authorities believe that nearly one hundred per cent of aneurysms are luetic. A rheumatic heart does not coincide with the clinical picture. We do not think the cardiac condition resulted from an acute rheumatic fever, but rather the arthritis mentioned we would associate with the metabolic disturbance.

A condition which could give us this venous pressure and also account for the symptoms is arteriovenous aneurysm. These conditions are not rare and to a certain extent some writers are reporting them in greater numbers. This condition cauld account for the high venous pressure. The other possibility is aortic reflux or leak. All authorities agree that a water hammer pulse must be present. We do not have this evidence.

The age of our patient is against congenital heart disease, in which a systolic murmur predominates. Forty-five to fifty years is perhaps a high period of life expectancy. She gives no history of curtailment of activities in childhood, shortness of breath, cyanosis, etc., existing throughout life, or periods prior to the present condition.

The spinal fluid has been rather mysterious,—straw colored; colloidal gold has been within normal range; only two red cells found in the fluid itself. We can not conceive of two red cells giving hemolysis and straw colored fluid. We believe we are able to explain this condition and connect it with our picture.

"Myxedema heart" has been recently reported in which all cardiac chambers are dilated and the electrocardiogram shows a complete absence of the T wave. Again, from the clinical evidence, this is presumably a small heart, not a dilated one. We do not believe this to be a fact.

Pericarditis with effusion is a strong possibility. The chest yielded only 80 c.c. of fluid. We are speculating whether that fluid was withdrawn from the pericardium rather than from the chest itself.

DR. C. R. K. SWETNAM

We have a very interesting case. If we had the time and the ability to go into it thoroughly, we would give you a treatise on medicine.

We have a woman sixty-five years of age, with a very well marked case of myxedema. There are some other things that do not seem to be entirely accounted for by myxedema; these we must account for and other conditions we must rule out. Evidently they were very suspicious of syphilis and did a lot of laboratory work. The blood Wassermann was negative, spinal fluid was drawn and examined

thoroughly. Wassermann and Hinton were both negative; the gold curve quite normal, which seems to rule out syphilis.

Examination of the blood shows that there was anemia. The history says "No marked achromia." I take that to mean there was a slight achromia. While some of the symptoms point to pernicious anemia, there were no blasts, and there is this slight achromia which should not be present in pernicious anemia. Color index was .9; usually in pernicious anemia the index is high, 1.0 or over. There is no stippling mentioned. One of the strongest things against pernicious anemia is the normal platelets. The platelets are one of the best guages in pernicious anemia, to know what stage it is in, and how it is progressnig. In pernicious anemia, the platelets are reduced, some change in size (likely to be large,) etc. It is true we have remissions in pernicious anemia, and this could be one from the blood picture, but not with the patient in such condition; failing very rapidly. We can not imagine anyone with pernicious anemia and such failure and still show normal platelets.

This anemia which our patient has would fit very nicely with myxedema.

The urine is negative except in amount. That would fit in with myxedema,—lack of water. The only other thing in the urine as shown by culture is colon bacillus, which is evidently a contamination.

The x-ray in this case is one of the most puzzling things. Something is pushing the heart to the right side and giving a very dense shadow. We immediately think of fluid. It may or may not be combined with very greatly thickened pleura. There are no symptoms in the history, increased temperature or pain, to indicate an acute condition in that chest, unless you would call filling with fluid acute. Subnormal temperature and no cough.

The spinal fluid is very interesting. The pressure is very slightly increased but not enough to make it pathological. They do not give us albumin or sugar test percentages but they do tell us that it is straw colored. Straw colored spinal fluid is not normal. What accounts for it? Straw colored, without cells and fluid being clear, not turbid, means it has been colored by hemolyzed blood. Hemolysis, we are told, does not take place for three days after hemorrhage occurs, increases gradually for fifteen days and then rapidly decreases. This yellow color is caused by the change of the hemoglobin into bilirubin, which is dissolved in the spinal fluid. We believe there must have been some hemorrhage within the dura in the last fifteen days. There are no focalizing symptoms; no history of nijury; nothing in the fluid to tell where it came from. It could be anywhere beneath the membrane or within the ventricles of the brain, but we believe it was due to congestion in the arachnoid. There is marked evidence of congestion to all organs, including the membranes surrounding the brain.

DR. JOHN W. FLINN

We had intended to go into detail in the discussion of myxedema but time will not permit. I will simply remind you that the outstanding feature in the disease called myxedema is decreased heat production, and this interference with metabolism is due to a pathologic condition in the thyroid gland. In the first place we will give you a definite diagnosis of myxedema and predict that the autopsy will show that many of the acini have been destroyed and that there is a good deal of proliferation of connective tissues and fibrosis in the thyroid gland.

Myxedema will not account for a great many things in this case. It will not account especially for venous engorgement nor for death at this time. Osler tells us that patients live ten to fifteen years and die of some intercurrent disease, very often tuberculosis.

We will tell you confidentially that there has been a great tendency for us to go off into the highways and byways in search of some other disease which would account for some of the symptoms more beautifully than the disease which we mention; but when we come to analyze this we can not find sufficient evidence to justify it. It would be a pretty thing to suppose enlargement of the thyroid gland, making pressure on the large vessels in the mediastinum and causing a great many of these symptoms. After giving a great deal of consideration to the case, we have come to the conclusion that the most logical explanation of this woman's venous engorgement and of her untimely death is hypertensive heart disease with hardening of the blood vessels. There is definite evidence of arteriosclerosis, definite evidence of hypertension, and we think these are sufficient to have caused the terminal picture.

For some considerable time authorities have noticed that there is a connection between myxedema and hypertrophy of the heart and myocarditis. Formerly it was supposed that this was simply coincidental, but as one writer has told us quite recently, the point has been made that there is a definite myxedematous heart, or a heart condition definitely due to a lack of thyroxin. Whether this is true or not, we do not know. Making allowances for all possibilities, we are not taking any chance in making a diagnosis of myxedema, but we are taking a chance in making a diagnosis of hypertensive heart with probable hypertrophy and dilatation in spite of the x-ray picture.

Our diagnosis is:

Myxedema;
Arterio-sclerosis;
Hypertrophy and dilatation of the heart;
Either hydrothorax or effusion;
Possible congestion of the uterus, the kidneys and the intestines.

The following discussion of this case was given before the Clinical Club of Phoenix, at their luncheon meeting of Dec. 9, 1929:—

DR. F. C. JORDAN

An American widow, aged 65, entered the hospital on May 13th, complaining of dizziness and slowness, and died six days later.

Four years ago, she had an attack of erysipelas and since, her skin has been puffy and pale; also, she has had increasing weakness and slowness. The symptoms coupled with dryness of the skin and hair, cold feet and hands, and a basal metabolism of minus 26, are found only when there is a hyposecretion of the thyroid. It is not uncommon for this condition to follow some severe infection like erysipelas. She has had three or four miscarriages at three months and one child died one day after birth. This would suggest syphilis, but a negative Wassermann with normal pulse and knee jerks would tend to rule out syphilis; yet, we must remember that brain syphilis does occur without many of the cardinal signs of syphilis. Thick and tortuous artery walls with a blood pressure of 195/110 denotes an arteriosclerosis which she probably had for some time as she was on a restricted diet for high blood pressure. Hypertensive heart disease usually causes death in one of two ways—either a cerebral hemorrhage or a gradual heart failure. The history is not typical of either although there are symptoms of cardiac distress in that she had had some dyspnea and edema of the legs which pitted on pressure. Edema caused by hypothyroidism does not pit on pres-

sure. Her blood picture is not abnormal. Is there any disease of the kidneys? She was in a stuperous condition—hard to rouse. Such symptoms could come from an impending uremia. There certainly is no evidence of any gross disease of the kidney. A specifc gravity of 1016 to 1026 showed the power of concentration; no albumen, no casts were found. This, together with a non-protein nitrogen of 25 would rule out uremia. In the lung there were no rales but flatness on percussion with diminished tactile fremitus and distant bronchial breathing in the left lung. X-ray of the left lung showed dence dullness except for a small area at the apex. The upper margin of this dullness was sharply defined and the heart and mediastinum were pushed to the right. Fluid in the left chest or a new growth in the left lung could push the heart to the right. A sharply defined line would indicate an effusion. You may get a pleural effusion with a new growth in the lung. This effusion, however, is nearly always bloody while the fluid removed in this case was of an opalescent yellowish color. There was cyanosis of the lips, hands and feet. This could be explained by an insufficient aeration of the blood in the lungs due to pressure of the fluid in the left chest; also, we must remember that ingestion of coal-tar products could produce cyanosis; also, any obstruction to the circulation of venous blood to the heart. The spade-like hands are quite common in myxedema. There was considerable calcification in the region of the pineal gland. I do not know what significance this might be. Spinal punctures showed a clear fluid with normal cell count but increased pressure. We are certain that she had arteriosclerosis and hypothyroidism, probable atrophy of the thyroid. We believe she had some brain lesion, whether it be brain syphilis or softening of the brain, we do not know. We do not think she had a meningitis. The normal temperature throughout her stay in the hospital would indicate that she did not have any infectious disease. Our diagnosis, therefore, is arteriosclerosis, atrophy of the thyroid, some softening of the brain, probably due to circulatory changes or minute hemorrhages. Fluid in the left chest, hypertensive heart changes without valvular lesion.

The following discussion of this case was given before the Clinico-Pathologic Conference at the Massachusetts General Hospital (see above journal):—

RICHARD C. CABOT, M. D.

Notes on the History

1. I do not see why erysipelas should make her eyelids puffy. I think something else besides erysipelas produced that change.

2. Why erysipelas is so much commoner in elderly people than it is in young people is a question of which one often thinks. I suppose the degree of immunity against streptococcus diminishes as we grow older. I remember vividly a man of seventy-three who had a hangnail on his thumb. It got a little red, a little sore. Otherwise he was well. Gradually an inflammation spread up his arm. There was nothing they could operate on. There was no pus. He had very big sore glands in his axilla, with high fever. He died within a week. At necropsy he had pus in every joint. What could you say about that except that the normal immunity against streptococcus which keeps us going in spite of our hangnails fails in old people, so that they are subject to erysipelas and other streptococcus infections?

3. That increasing weakness and slowness in movement is not surprising at her age.

4. What is the history man thinking of when he records dry hair and poor circulation in the hands and feet?

A Student: Myxedema.

Dr. Cabot: That is a rare disease, so I am skeptical, but still it is possible.

There are other causes than myxedema for loss of hair. At sixty-five it is not surprising.

5. Dizziness after quick motions of the head is very common again after sixey. Generally this is said to be due to arteriosclerosis. We do not know it.

6. Her not sleeping well is not remarkable. All that we might call growing old—that is, it might be nothing but that. I suppose growing old means arteriosclerosis in the great majority of cases.

7. "Spade-like" is one of the medical slang terms, rather melodramatic. I have never seen a hand that looked like a spade. Certainly no patient would say it. It is a term that is often associated with myxedema and other diseases, but I think it is not a good descriptive term.

8. The man taking this history had the intelligence to say that the patient was exposed to tuberculosis; but still at sixty-five it is not very important. It would be very hard luck if she got as far as sixty-five and then caught it.

9. The history of miscarriages makes us think of syphilis. It does not prove it.

10. High blood pressure is the most definite thing in the history. It has been almost colorless up to that point. She was evidently told that she had high blood pressure, and that is significant. Nothing else is helpful for diagnosis. So far as we have got, the lead is hypertension and the heart troubles that go with it.

Notes on the Physical Examination

1. The first few statements in the physical examination suggest myxedema, but it is perfectly possible that they are simply old age plus edema.

2. "Superabundance of tissue over the clavicles and upper borders of trapezius muscles." They are thinking of myxedema. But in an obese woman, as she is stated to be, that is very hard to interpret as indicating anything important.

3. In the description of the tongue again they are thinking of myxedema.

4. The percussion measurements are probably not true. They mean from the midsternal line, and of course 5.5 is not nearly enough.

5. The heart may be displaced, but we should not say so without an x-ray.

6. In the absence of cerebral lesions the blood pressure shows that we certainly have hypertension.

7. These lung signs are equivocal. They do not fit exactly with fluid or solid. We have bronchial breathing, flatness and increased whispered voice, all in favor of solidified lung. We have diminished tactile fremitus, which is in favor of fluid or high diaphragm with the spleen or the liver sucked up into that area. That on the whole seems to me the more likely, that is, the subdiaphragmatic organs pushed up into the chest, but that does not account for the bronchial breathing. I do not account for that. It might be due to this subdiaphragmatic mass.

8. They evidently think the heart is pulled or pushed or drawn over to the right by something, and we need very much to know what that is. So far I have no idea.

9. The absence of pulsation in the dorsalis pedis arteries does not mean that they are obstructed. We often cannot feel them in old people. We do not need to suppose any local disease.

10. The fact that there are hemorrhages in the fundi is of course an important point.

11. Colon bacillus in the urine culture does not mean much in a case like this. We get a good many of those cultures.

12. The blood shows a moderate presumably secondary anemia, though there is no marked achromia.

13. The basal metabolic rate, minus 26, obliges us to consider myxedema seriously, though in a person as near death as this there might be a good many other things that would produce some lowering.

14. We have no good reason to suspect brain tumor. There has not been much to suggest it anywhere.

15. Eighty cubic centimeters is very little pleural fluid if they took all they could get. It is not nearly so much as we should expect.

16. The red count is a million more than last time. It makes us believe that one of the counts is wrong. But the latter might have been due to surface cyanosis and not a typical red count at all.

Differential Diagnosis

I am much in the dark. The x-ray is our chief evidence. We have a lowered basal metabolism and other points in the history that go perfectly well with myxedema. Could she have died of myxedema? I never knew of anyone's dying of myxedema. Those that have been nearest to death were cases with a very severe secondary anemia. If she had myxedema I do not think it is important in relation to the cause of death. She died I think of something that affected her chest, especially her left chest, and that has to do with her heart's being over on the right. What can that be? Can it be pleurisy? Why not?

Students: She had no temperature—no pain—no friction rub.

Dr. Cabot: The chief point is that nobody ever died of pleurisy. We know she died. Therefore she did not die of pleurisy as anything important in her case. The other points against it are also perfectly good. She had no fever. The fluid would have been larger in amount. There certainly would have been more than eighty cubic centimeters. What else could it be?

A Student: Aneurysm.

Dr. Cabot: That I suppose must be thought of. What else?

A Student: Arteriosclerotic heart disease with passive congestion.

Dr. Cabot: That would not account for displacement of the heart.

A Student: Lung abscess.

Dr. Cabot: That is a possibility. The difficulty is that we have no fever, we have no characteristic sputum, and lung abscess does not displace the heart.

A Student: Tuberculosis.

Dr. Cabot: She is obese and sixty-five. Those two points are pretty strong against tuberculosis, but they do not exclude it. She may have had tuberculosis, but I do not see how she could have had it in a form that would displace the heart. When it displaces the heart it is on the side toward which the heart goes, unless the heart is displaced by tuberculous pneumothorax. Then it would go over to the other side. But we have no evidence of pneumothorax so far as I see. That sharp border of the x-ray shadow is not like a fluid level. It is not horizontal. Neither does it slope up to the upper corner. That is not the usual shape for a fluid level. Going back to tuberculosis, if she had tuberculosis on the right side where the heart is pulled over as by adhesions, it ought to show there. If she had it on the left side it would be at the base of the lung, a rare place. She had no fever, no sputum, and she died. I do not believe it is tuberculosis. What else could it be?

A Student: Something in that left upper quadrant in the abdomen pushing her heart over.

Dr. Cabot: I thought of that at first. But we have nothing to show where the diaphragm is on the left. It is not a clear outline for a high diaphragm. What could we have in that region which would push the diaphragm up and displace the heart that much? It could be an enormous spleen. But if it were an enlarged spleen it would be down to the umbilicus. Or could it be a kidney? Yes, as far as I know. Then it would have to be malignant disease. If it were malignant disease it might not show in the urine. It certainly does not show. The common tumor of the kidney, hypernephroma, ordinarily has metastases before death. In this case we had an x-ray of the skull as well as the rest and we should expect to find some of those metastases. Then I have never known death from tumor of the kidney without a great deal more anemia than this. My difficulty therefore in supposing something below the diaphragm is that I cannot see what it could be. I do not see how it could be spleen or kidney or any other organ. Disease in the intestine could not do it without more functional disturbance.

A Student: How about infarct of the lung?

Dr. Cabot: Infarction of the lung of course has to have a cause, and we generally have some hint of that cause. It usually comes from heart disease. We have no evidence of that. She has a hypertension, but very little to convince us that she has back pressure and chronic passive congestion such as you get with infarction of the lung.

Could it be a massive collapse of the left lung? No. The heart is going the wrong way.

A Student: Syphilis of the lung.

Dr. Cabot: We have never made the diagnosis right in this hospital.

A Student: How about parasitic infection?

Dr. Cabot: We do not know where she lived. It might have been that if she lived in South America.

Miss Painter: She was born in Massachusetts and had lived here most of the time.

Dr. Cabot: That is a good point against parasitic infection. I do not think we need to consider that.

So I come down to malignant disease, epithelioma or endothelioma of the right pleura. I do not know how to exclude that. It is true that that usually gives bloody fluid in the tap, but not always. I remember several cases where I myself tapped and got clear fluid. I cannot rule it out. The possibility of something in the glands of the chest must be remembered, as the cyanosis of the upper part of the body certainly suggests pressure on the venous trunks and the cardiac displacement could come from compressed bronchus from metastasis or enlarged glands.

A Student: Could a malignant tumor of the pluera account for all the various symptoms and signs that you referred to a little while ago in connection with myxedema?

Dr. Cabot: I do not believe it could. I think we have to make a separate diagnosis.

A Student: What is the frequency of such metastasis? Is it common in the local glands or in the thyroid?

Dr. Cabot: I never heard of it in the thyroid. Certainly most of the metastases from the lung or pleura are local, regional.

A Student: What happens to the metabolic rate in malignancy?

Dr. Cabot: Except in leukemia it is low ordinarily. But I never knew it to be so low as minus twenty-six.

A Student: Might not the palpable mass in the liver be metastasis?

Dr. Cabot: Yes, perfectly well. It is drawn in a suggestive way in the diagram.

A Student: Was there any sediment test on the chest fluid?

Dr. Cabot: Have we all the data?

Miss Painter: The record breaks off unfinished.

Dr. Tracy B. Mallory: The findings were those of a transudate rather than an exudate.

Dr. Cabot: To sum up, I see three factors,—certainly two, and I believe three. (1) I believe she died of malignant disease in the chest starting either in the pleura or in the lung, I do not know which, with metastatic glands accounting for cyanosis in the upper part of the body. (2) Hypertension, which could not be accounted for by this. It might be attributed to weakened heart and this strikingly low metabolic rate, which I do not know how to account for unless by (3) myxedema. I should say we have evidence of three factors, two of which had nothing to do with her death and one of which was the central cause.

A Student: Isn't it possible that the hypertension was a mistake? The second record is 145/100, which is a good deal less than 195.

Dr. Cabot: I do not know any special reason to suspect that it is a mistake. You often get as much variation as that near the end of life. The second one was presumably at the end of life. Have we the dates of these blood pressures?

Miss Painter: On May 13 it was 195/110. On the next day it was 145/100. On the 16th it was 180/114.

Dr. Cabot: I think we have to say it was hypertension.

Interpretation of x-ray: Probably fluid in the left lung field. Possibly a mediastinal mass.

Clinical Diagnosis (From Hospital Record)

Myxedema.
Hydrothorax.
Cardiac failure.

Dr. Richard C. Cabot's Diagnosis

Malignant disease primary in the lung or pleura, with metastatic glands.
Hypertension.
Myxedema.

DR. TRACY B. MALLORY
Anatomic Diagnosis

1. Primary disease.
   Myxedema.
   Arteriosclerosis.
2. Secondary or terminal lesions.
   Arteriosclerotic heart disease.

Hypertrophy and dilatation of the heart.
Hydropericardium.
Hydrothorax (bilateral).
Arteriosclerotic nephritis (benign type).

Dr. Malory: I can confirm two diagnoses in this case, but not the third. The patient did have myxedema. The total weight of the thyroid gland was six grams, as against a normal of between 25 and 30. It consisted almost purely of scar tissue in which only very rare parenchymal cells could be identified. She had an extreme degree of arteriosclerosis, and I think the other findings are all secondary to that.

The chest contained over three liters of fluid, approximately 1000 cubic centimeters of fluid in each of the pleural cavities, and a little over 1000 in the pericardium. In view of the x-ray findings much of this must have accumulated very rapidly in the last few hours of life. The lungs were almost completely atelectatic as a result of pressure from the large amount of fluid.

The heart was much enlarged, weighing 550 grams. There was marked sclerosis of the coronary arteries, without however any marked narrowing of their lumen. The splenic artery showed an extreme tortuosity, the type of arteriosclerotic change that is properly described as a cirsoid aneurysm.

The kidneys showed a slight degree of vascular nephritis, which is a common finding in cases with long-standing hypertension.

A Student: How about the liver and gall-bladder?

Dr. Mallory: They were quite negative except for slight passive congestion.

Dr. Cabot: What clinical diagnosis was made?

Dr. Mallory: Myxedema and cardiac failure. The fluid in the pericardium was never suspected.

A Student: Where did the fluid come from?

Dr. Cabot: It was dropsical.

I think they knew in the wards a good many things they did not tell us. There is nothing in the record to suggest cardiac failure. We got no idea of cardiac failure or of dropsy anywhere. I think the heart must have shown evidence of weakness which we did not get in the record.

I was certainly thrown off when they got only 80 cubic centimeters of fluid out of 1000 cubic centimeters actually there. Although we made a clean miss of diagnosis, I do not know how to blame myself or you for the mistake.

Was the heart displaced?

Dr. Mallory: No, I do not think it was. The dullness was due to that big pericardium without much question, and the signs in the lung were due, I suppose, to atelectasis. I think the death was immediately due to asphyxia from insufficiently aerated lung.

Dr. Cabot: This is a very rare kind of death. Ordinarily we take suffocating chest fluid out. Why they did not do it here I do not know.

GENERAL GLANDULAR ENLARGEMENT LASTING THIRTY YEARS OR MORE

(Case 14421, Case Records of the Massachusetts General Hospital, from New Eng. Jour. of Med., of Dec. 6, 1928, p. 1167.)

CASE HISTORY

An American stationary fireman forty-three years old entered September 12 through the Emergency Ward for study of upper abdominal pain and enlarged glands. He was semistuporous and very irritable.

Ever since he was a small child he had had enlarged glands in his neck and axillae. They did not change from childhood until after he was grown up. Then at times they increased, occasionally reaching the size of a large walnut and remaining so for a few days, then going down again and not troubling him for months. He had been in fairly good health until September 1, although for several years he had had epigastric distress and eructations of gas after heavy meals. Seven months before admission the inguinal glands became enlarged after some hard lifting. These glands at first were tender for a day or two. The glands in the neck and axillae had never been tender. In May his feet and ankles began to swell during the day, reaching two or three times their normal size after he had been on his feet for a long time but returning to normal overnight. At this time there was more marked enlargement of the inguinal glands. Under treatment by a physician the edema disappeared. He had urinated once or twice a night since that time. The night of September 1 after eating a few pieces of chocolate candy he was suddenly seized with severe colicky epi-

gastric pain which lasted for over an hour and gradually turned into a gnawing dull ache which kept him awake all night and for the five following nights. After this attack he ate very little food because it seemed to cause gas and increase the discomfort. Soda gave no relief. The pain came in waves. When he had attacks of sharp pain it seemed to run around to the lumbosacral region. Four weeks before admission he had two or three attacks of shooting and darting pain all through his legs.

His bowels had always been constipated. He had slight dyspnea and palpitation on exertion. For a pear he had occasionally urinated at night. There was some question as to girdle and shooting pains. Ten years before admission he weighed 215 pounds, He thought he had lost some weight in the past six months.

His mother, who died of cancer of the breast, had some "heart failure." His four children had enlarged glands, not so marked as his own.

Clinical examination showed a man with obvious loss of weight and marked pallor, cooperative but drowsy. Moderate pyorrhea. Nasal septum deviated to the left. Left submaxillary and supraclavicular and right cervical glands enlarged, measuring one-half to three centimeters. In both axillae and groins were glands the size of a peach stone, the inguinals firm, all others moderately soft. Marked flaring of the lower costal margins. Harrison's groove. Diaphragmatic excursion limited. Inspiratory wheeze on the left, front and back. Apex impulse of the heart not seen or felt. No enlargement to percussion except slight increase in supracardiac dullness to the left. (Total 6.5 centimeters.) Pulse and artery walls normal. Blood pressure 115/70 to 96/65. Abdomen slightly distended, with tympany in the center and shifting dullness in the flanks. No fluid wave made out. Veins prominent over the upper abdomen. Liver dullness increased downward to five centimeters below the costal margin. Edge not felt. In the left upper quadrant a mass, probably the spleen, descended five centimeters below the costal margin on inspiration. Right pupil greater than left; both regular and reacted normally. Fundi: discs slightly pale, no exudate or hemorrhage. Genitals, extremities and reflexes normal.

Urine: 30 to 55 ounces, red at three of five examinations, specific gravity 1.020 to 1.030, a slight trace of albumin once, bile twice, rare to occasional leukocytes at three of five sediment examinations. Blood: 3,600 to 8,400 leukocytes, 61 to 100 per cent polymorphonuclears, hemoglobin 75 to 45 per cent, reds 4,500,000 to 3,250,000, moderate to marked achromia, moderate to slight poikilocytosis in all of five smears, anisocytosis in the last two, platelets always reduced. In one smear several of the very pale red cells showed rings in centers made up of very small granules, possibly artefacts or Cabot's rings. Two Hinton tests negative. Non-protein nitrogen 27 milligrams. Icteric index September 13, 3, October 5, 40, October 7, 20. Stools negative.

Temperature 96.8° by mouth to 103°. Pulse 80 to 140. Respirations 20 to 44.

X-ray showed the hilus shadows markedly enlarged in size. There was a lobulated mass in this region. The interlobar septum was visible on the right. There was fine mottled dullness throughout both lung fields. The changes were most marked in the upper lobe. The pelvis and lumbosacral spine showed no abnormalities. Re-examination October 9 showed no definite change since the previous observation.

September 17 a gland was removed from the neck for examination. September 19 x-ray treatment was begun.

The patient had some pain. From September 15 to 19 he had no movement of the bowels. The temperature was 101° to 103°. There were medium rales at both bases with diminished breath and voice sounds and with a questionable friction rub below the angle of the right scapula. The patient grew rapidly weaker, thinner and paler and dozed much of the time. The glands diminished slightly in size. The mass in the right upper quadrant grew much smaller. He had no appetite and took only milk and a few crackers. His mouth was dry, his tongue shiny and stiff. October 6 there were numerous fine to medium rales in both backs and a few coarse crackles at the left base. Fluid persisted in the abdomen. The glands diminished markedly in size. October 6 the spleen could not be felt. The leukocyte count was 6,000. The blood contained no lymphocytes. He continued to fail rapidly. Rales increased in both sides of the chest, more marked on the right. From October 4 to 10 the temperature was never below 100.7° rectal or above 102°. The pulse was 100 to 140, the respiratory rate 23 to 40. There was moderate pitting edema of the feet. October 10 he died.

The following discussions of this case were given before the Phoenix Clinical Club at their luncheon meeting on Dec. 9, 1929:—

DR. FRED G. HOLMES

The enlargement of the lymph nodes of the neck and axilla from early childhood may have been part of childhood rickets and have little bearing on the case. The whole picture changed some seven months or more previous to his death when the chain of symptoms surrounding the lymph nodes came to the fore.

Tuberculosis of the lymph nodes in a man of 43 years with the course as shown in this case may be excluded at once.

The differential diagnosis would seem to lie between Hodgkin's disease, pseudoleukemia and lymphosarcoma. In the study of this case, we are in the position of the clinicians handling it; we will probably need the findings on the gland removed from the neck for a definite diagnosis.

We may, however, venture some likely guesses. Hodgkin's is much more likely to occur at an earlier age than in this case, whereas pseudoleukemia is likely to occur at this ago, while lymphosarcoma occurs most commonly between 25 and 55 years. The blood picture would fit better with Hodgkin's disease or lymphosarcoma than pseudoleukemia, as, in hte latter, there is usually an increase in the relative lymphocyte count even though the total leucocyte count may not be elevated, while here we have almost a complete absence of lymphocytes. The remissions in the size of the lymph nodes and remissions in the symptoms are quite characteristic of Hodgkin's disease. It would appear from the description that the glands involved are a little more numerous and there is hardly enough periglandular infiltration of surrounding tissues to indicate lymphosarcoma.

This would appear to favor the diagnosis of Hodgkin's or pseudoleukemia. The anemia, enlargement of the liver and spleen, edema and ascites, temperature, pains probably due to pressure of the enlarged lymph nodes, respiratory symptoms due to enlarged glands, are all symptoms which might be found under certain circumstances in cases of Hodgkin's, pseudoleukemia or lymphosarcoma.

The diminution in size of the glands under x-ray therapy would occur with either condition, although lymphosarcoma shows the most marked reduction, the glands melting away and the patient soon following. It seems the weight of the evidence here is in favor of Hodgkin's disease, considering the blood

picture, the glands involved, and the remission in the disease.

He may have had a terminal pneumonia.

DR. ORVILLE H. BROWN

An analysis of the signs and symptoms of this case are as follows: Male; age 43; fireman of stationary engine. As a small child he had enlarged lymph nodes in the neck and axillae; at times they would increase to the size of a walnut for a few days, then recede. For several years before admission he had had epigastric disturbances and complained of gas after eating. Seven months before admission inguinal glands enlarged. Four months before admission the feet began to swell. Eleven days before admission he had severe colic and epigastric pain after eating chocolates; a dull ache lasted five days. Shooting pains developed at times in his legs. He had had dyspnea and palpitation on exertion. He has always been constipated. He had lost 25 pounds in weight in the last 24 years up to one year ago and thinks he has lost since. His four children all have enlarged lymph nodes.

Examination shows obvious loss of weight; marked pallor; drowsiness; pyorrhea; deviated septum; lymph nodes of neck measuring one-half to three cm. in diameter; those in the axillae and groins are as large as peach stones. Inspiratory wheezes exist in the left lung. The abdomen is distended and shows dullness in the flanks. The sides of the abdomen are prominent. The liver dullness is increased 5 cm. downward. The spleen is evidently enlarged five cm. below the costal margin. The right pupil is larger than the left. The following were laboratory findings: Bile in urine; low leucocyte count in blood; marked increase in per cent of polys; anemia; achromia; poikilocytosis; anisocytosis; reduced platelets; icteric index increased. Temperature, pulse and respiration were variable; lobulated masses were in the mediastinum; increased shadows existed in the hyla. X-ray treatments were given. Obstipation developed in hospital. Patient grew worse in every way, although the nodes diminished in size. Fluid persisted in the abdomen. On October 6th there were no lymphocytes in the blood. Rales were in both chests. Pitting edema of the feet existed at times.

The signs and symptoms of this case point conclusively to and can be explained by pathology in the lymphatic system. There had been lymph node enlargement since childhood. During adulthood the glands had swollen and receded. Just how often this occurred is not recorded. It is a particularly interesting point in the history and should have received more investigation.

The first question to answer in this case is in regard to the enlargement of the lymph nodes which existed in childhood. It is well to know in this connection that his four children had lymph node enlargement at the time of his acute illness. Lymph node enlargement is not ordinarily a familial disease.

The fact that the lymph nodes of the axillae were enlarged in this case means more than had only those of the cervical region been enlarged. Cervical lymph nodes are commonly enlarged from infections of nose, throat and mouth.

Lymph node enlargement in childhood may come from about the same set of causes as lymph node enlargement in later life. Therefore it is necessary to discuss here the various causes of lymph node enlargement.

The knowledge of lymph node disease is chaotic. There are, however, certain more or less definite classifications such as Hodgkin's disease, status lymphaticus, glandular fever or mononucleosis, simple adenitis, tuberculous adenitis, syphilitic adenitis, Gaucher's disease and indefinite classifications variously known as granuloma, lymphogranuloma, lymphoma, lymphoblastoma, lymphosarcoma, aleukemia, lymphatic leukemia, etc.

There are a few points in the physiology of the lymphatic system which are of particular interest.

Rous has shown that, at least in the dog, 150 to 200 per cent of the lymphocytes in the blood stream at any one time are discharged from the thoracic duct in twenty-four hours. The lymphocyte is apparently an "end cell." It has been definitely shown that the lymphocytes play an important part in immune reactions to certain types of infection, such as tuberculosis, cancer and syphilis. Lymphotoxic disease such as typhoid, tuberculosis and Hodgkin's disease, are notably diseases of youth. Lymph tissue is more prominent, and seems to play a greater part, in the life of youth than of the adult. It tends to atrophy with age.

An unexplained and yet interesting phenomenon is that the male is far more susceptible than the female to most of the diseases which affect the lymphatic tissues.

The structure of the normal lymph node should be known in order to understand the diseased lymph node. It is usually spherical to oval and shaped like a bean. It has a convex surface. The blood supply enters through indentations known as the hila. The nodes have: (1) A capsule, mainly connective tissue; (2) trabeculae with lymph sinuses extending from the capsule into the medulla, where they anastomose and continue to the hila; (3) lymphocytes in the cortex in rounded masses or follicles; (4) lymphocytes in the medulla in elongated masses or lymph cords; (5) a fine framework of reticular itssue supporting the entire structure; and (6) lymph vessels leading from the capsule and entering at the hila.

Hodgkin's disease is probably the outstanding disease of the lymphatic system. It occurs twice as often in the male as in the female. It is a disease primarily of youth although cases are on record of the sixth decade. After the third decade, however, the chances of having Hodgkin's disease rapidly diminish. The greatest number of cases occur between the ages of fifteen and twenty-five. There is relatively little proven of the etiology of Hodgkin's disease. A number of workers have isolated diphtheroid bacilli pleomorphic in character, from numerous cases. The organism has been isolated also from other diseases. In a few instances in animals the injection of cultures of this organism produced lymph node enlargement. This organism is not accepted as yet as the etiologic agent. There is a general feeling that Hodgkin's disease is a result of infection the nature of which is yet to be discovered. The pathology of the lymph nodes in Hodgkin's disease is apparently definite. The characteristics of the node of Hodgkin's are as follows: (1) The capsule is thickened; (2) there is proliferation of the reticulo-endothelial cells; (3) the trabeculae have disappeared; (4) there is a hyperplasia of the lymphatic tissue especially in the medulla; (5) the cortex and the medulla both become over filled with lymphocyte, plasma, eosinophile, cosmophile, epitheloid and multinuclear cells. (The multinuclear giant cells are known as the Dorothy Reed cells and are the characteristic cells of Hodgkin's disease); (6) the lymph channels of the nodes are filled with cells and are therefore practically obstructed; and (7) the reticular connective tissue is hypertrophied. The nodes do not caseate. They may be either soft or hard. There are usually one or more groups enlarged and they get very large. The disease may occur in any node or group of nodes within the body. It may be on one side or it may be on both sides.

It may be in limited areas or general. The respiration, heart and other important functions may be seriously interfered with through pressure. The course of the disease is invariably progressive. Death may ensue in six to ten months or even sooner. It is rarely longer than three to four years. A rare case or two has lasted eight years. The treatment seems to be of little avail but a few cases have been benefited by rational, radical measures, such as elimination of all foci of infection and extensive x-ray therapy.

Status lymphaticus is instability of the lymphoid tissue, in which the tonsils, Peyer's patches, the surrounding follicles and the Malpighian bodies of the spleen are hyperplastic; the lymph nodes also may be palpably enlarged. The thymus is invariably enlarged. The figure of the affected individual is characteristic; the muscular system is well developed; the skin is delicate in texture, clear and pink; the male is female in configuration; axillary hair is thin; the female type of pubic hair exists and all in all the body is gracefully constructed.

Mononucleosis is an acute condition of childhood, in which the cervical nodes are considerably enlarged for ten dyas to two weeks or even longer. Recovery takes place in a short time.

Simple adenitis of the lymph nodes may be acute or chronic—usually regional from drainage of infected areas. It usually comes from a discoverable etiologic factor. In the chronic cases there is a chronic cause and the adenitis may be more general.

Tuberculous adenitis is comparable to simple adenitis. The nodes which drain areas affected with tuberculosis may be greatly enlarged and the affected nodes are prone to caseate. Ordinarily surface nodes fuse together making a cold abscess and perhaps a draining sinus. The pathology of tuberculosis of the lymph nodes is the same as the pathology of tuberculosis elsewhere except as modified by the structure of the nodes.

Syphilitic adenitis of the lymph nodes is common in syphilis and occurs in all stages of the infection. It has a tendency to become chronic, lasting a number of months or years and usually ends in a fibrosis or atrophy of the nodes. The nodes are usually small except those draining the area bearing the chancre.

Gummata may affect lymph nodes and cause numerous enlargements, but ordinarily this occurs in but a few nodes. We know syphilis to be a chronic disease and one which is transmitted from parents to children.

Gaucher's disease is primarily of the spleen—a familial splenomegaly in which several children of one family usually are affected. The lymph nodes show a variable amount of involvement though generally not more than palpable. The spleen is usually much enlarged, although it maintains its regular shape and normal appearance, often weighing as much as seven pounds. The liver is generally enlarged, showing a marked increase in interstitial connective tissue. This disease occurs more commonly in the female than in the male. It usually occurs in the sisters of one family and of one generation. There is no instance on record of transmission from parent to children. Leukopenia develops as an early persistent factor. Hemorrhages of the skin and yellowish discoloration are common.

The illy defined conditions variously known as lymphoblastic leukemia, lymphatic leukemia, pseudoleukemia, lymphoblastic aleukemia, lymphosarcoma, lymphoma, benign lymphoma and malignant neoplasms are poorly understood; they seem to be related and yet extreme examples of one or another of the classifications give evidence of definite entities.

Dr. Nellis B. Foster describes a type of lymph node enlargement under the head of lymphoblastic aleukemia and leukemia. This is a progressive lymph node enlargement with hyperplasia of lymphatic tissue with or without a leukemic blood picture. It seems to be closely related to Hodgkin's disease. There is a tendency for a general lymph node enlargement. Lymphocytes of the small type are lacking in the glandular section. There is an extensive proliferation of lymphoblasts without differentiation; the acitvity of the nodes is due to the complete infiltration and the filling of the spaces and a thinning of the reticulum. The blood shows a marked reduction of small lymphocytes in the aleukemia form. There is no increase of the large mononuclears. In certain cases the aleukemic type may develop into the leukemic type.

Numerous writers have used the term lymphosarcoma. Virchow originally applied this term to Hodgkin's disease and to pseudoleukemia. Kundrat used the term to designate a type of disease closely simulating sarcoma of other organs. There is a considerable percentage of men who consider it related to Hodgkin's disease. It does not start in a single node but in a group of them. It has a tendency to invade important structures and even metastasize through the blood stream. The new growth consists of reticular tissue and lymphoid cells usually of the large type. The cells may infiltrate surrounding tissue but unlike sarcoma there is no destruction of the invaded tissues. The symptoms depend upon the location of the disease. In certain cases a leukemic blood picture has developed during the course of the disease. The disease is uniformly fatal, usually in less time than Hodgkin's disease.

Benign lymphoma was described by Senn as "a tumor formed of lymphoid tissue produced by a matrix of lymphoblasts." These are usually single and of more pathological interest than clinical. They last a long time.

Malignant neoplasm may arise from lymph nodes. They may be fibrosarcoma or of the round cell variety. The growth and development and metastases are not unlike sarcoma arising in other organs but are in contrast to the group of lymph nodes described as lymphosarcoma according to Foster. Some authors find it difficult to distinguish between this type of growth and the lymphsarcoma and Hodgkin's disease.

The case we have for study does not fit accurately into any of the diagnostic classifications which we have presented. We have here a man who for thirty years had lymph node enlargement. The facts about Hodgkin's disease practically rule this case out of the diagnosis, unless the Hodgkin's disease came on top of a chronic lymph node enlargement from other cause.

The diagnosis of Hodgkin's disease could only be made by the histological picture of a slightly enlarged node. The latter stage of the case might fit into the classification of Hodgkin's disease. The glands, however, are not as large as ordinarily occur in Hodgkin's disease.

I think we may safely say that the case does not belong in the classifications of tuberculosis or syphilis or mononucleosis; nor does it seem to be Gaucher's disease, notwithstanding the enlarged liver and spleen.

Probably it is not a malignant neoplasm according to the definition of Foster. It probably is not a lymphosarcoma according to the definition of Kundrat.

It seems to fall fairly well into the classification of lymphoblastic aleukemia or pseudoleukemia. The fact that there were few or no lymphocytes in the blood stream suggest total or nearly total destruc-

tion of the lymphocyte producing tissues of the body.

The diagnosis then in this case appears to be lymphoblastic aleukemia, or lymphoblastoma or pseudoleukemia,—chronic for many years with recent acute exacerbation; enlargement of the liver and spleen associated with the lymphoblastoma; edema of the legs—from pressure exerted by the enlarged nodes; gastric disturbances—also secondary to pressure; cardiac dilation; dyspnea from heart strain and from mediastinal tumor pressure; pyorrhea; deflected septum of the nose; asthmatic condition of the left lung secondary to pressure; ascites; inequality of the pupils from pressure; lymphopenia; reduced platelets and anemia as changes of blood forming organs; and obstipation also secondary to pressure from the increase in size of the lymphatic tissues.

The following discussion was given before the Yavapai County Medical Society and Medical Officers of Fort Whipple, at their meeting of December 10, 1929:—

DR. G. O. BASSETT

This is a case marked essentially by two factors, a moderately severe anemia and a generalized glandular enlargement. He dies of terminal pneumonia after a month's rather acute illness. The majority of cases of Hodgkin's disease are prolonged over a period of months rather than weeks.

Let us for a moment go over the blood picture again. Let me show you just how difficult it is; even though we can narrow the condition down to the relatively small group of diseases included in the generalized lymph adenomata pseudoleukemias and lymphosarcomas. The blood picture of Hodgkin's or pseudoleukemia shows a relative leukocytosis, sometimes as high as 30,000 or more, which might be confused with the picture of leukemia. In this case it is never higher than 8,400; polys from 61 to 100 per cent, which is even more confusing in that lymphocytes are not mentioned. In fact, in the last report, no lymphocytes were found in a white count of 6.000. In Hodgkin's disease it is not unusual to find the leukocyte count around normal, although it may go up relatively high, but there is a general tendency for lymphocytes to be present and to increase. There is no eosinophilia and that, as acknowledged by all authorities, is one of the few characteristic features of Hodgkin's. Platelets in this case show nothing unusual,—in Hodgkin's they usually increase. We have to rule out Hodgkin's disease unless we fall back on the possibility that this is an atypical case.

The group of diseases of more malignant nature, classified under the heading of lymphosarcomas, fit this case more accurately in some respects than does Hodgkin's. Most writers state that lymphosarcoma is a more fulminating type of Hodgkin's disease and shows less change from the normal blood picture than does true Hodgkin's. The blood picture may be absolutely blank. The tendency in all malignancies is to show, terminally, some leukocytosis. We have in lymphosarcoma, as in all malignant conditions, a relative anemia which gradually increases, which is true in this case. Eosinophilia and increased platelets are not necessary to point to lymphosarcoma.

The general features point more definitely to lymphosarcoma than to Hodgkin's disease. In the first place, it is a rather fulminating thing and he was sick only about a month. The man is cachectic, which is very apt to occur in all malignancies. The history tells us there was fluid in the abdomen, edema of the feet, and possibly other places. Although it is possible in the last stages of Hodgkin's to have fluid in the extremities, it is more likely to appear in malignant conditions, especially lymphosarcoma.

X-ray of chest shows a large mass involving the mediastinal structures. That x-ray is very interesting. We had, of course, to deal primarily with tuberculous involvement of the glands in childhood. It is always probable later on that tuberculosis may be a factor in any condition that develops. The x-ray reading, "mottled dulness of both upper lobes," probably means mottled appearance of the lungs and dulness on percussion. Lymphosarcoma has a habit of pushing out into the lung and could give a picture somewhat resembling this. Gastro-intestinal symptoms we think are due to pressure and lymph node enlargement. The symptoms, all the way through, could be covered by a diagnosis of generalized adenomatous condition of this man's body, enlarged lymph glands which are a process of the rapidly developing lymphosarcoma.

Our diagnosis is lymphosarcoma, although it could easily be Hodgkin's disease; the possibility of tuberculosis as a contributing factor can not be ruled out; and terminal pneumonia.

The following discussions of this case were given at the Clinico-Pathological Conference at the Massachusetts General Hospital (see above journal):—

RICHARD C. CABOT, M.D.

Notes on The History

1. This is a very unusual history, if true. One sees plenty of people who have enlarged glands, but not for many years like this with retention of very fair health. I do not remember any case exactly like this and I can make no special conclusion from that statement. They do not sound like leukemia or Hodgkin's or syphilis or tuberculosis, the ordinary four cases of enlarged glands.

2. The pain was peristaltic presumably.

3. I am very much in the dark. Epigastric pain of course is ordinarily due to gall-stones or peptic ulcer or simple things like constipation, but the rest of the symptoms do not back up any of these causes and so far I have nothing of importance to say.

Notes on the Physical Examination

1. The pain in his legs made them think of the lightning pains of tabes. Tabes is a cause, though not a common cause, of epigastric pain. But they do not seem to be clear on that point in his history.

2. Three centimeters diameter means a sizable gland. Half a centimeter is a size that half the the normal people have in neck, axillae and groin, but three centimeters is something. So far the enlarged glands are the essential lesion that we have.

3. There is ascites. The prominent veins make us wonder whether cirrhosis of the liver or some other less common form of portal stasis is the cause of this ascites.

So far we have enlarged glands, big spleen, fluid in the abdomen, enlarged veins in the epigastrium, perhaps enlarged liver.

4. I have never seen 100 per cent of polynuclears in any blood. I do not believe it. But of course they get up into the nineties quite often. This blood shows secondary anemia with polynuclear leukocytosis; it is not characteristic of any blood disease; it is consistent with Hodgkin's, consistent with a syphilis or with a cirrhosis of the liver.

5. Only one of the x-ray plates, the earliest, is a good plate according to my standards. I suppose as he got sicker it was harder to take them. It certainly looks as if the masses around the hilus of the lung were larger than they ought to be and as if there was more than normal spotting throughout both lungs. There are no sizable nodules such

as we get in metastatic malignant disease. I do not see much difference between September 12 and October 8, but one cannot be sure about the shadows because everything is so dim.

We have here, then, something that can perfectly well be Hodgkin's (lymphoma), but we are not forced to it in any way. It is not yet characteristic or clear.

6. Presumably when the gland was taken out they found out what the diagnosis was, but they do not tell us. When we have a superficial gland there is no use waiting; we can have an antemortem examination.

7. They no longer have any doubt about the fluid in the abdomen on October 6. They seemed a little doubtful about it before.

8. They are very definite in the statement "no lymphocytes". I wish we had the slide.

A Student: Wouldn't that be consistent with x-ray treatment?

Dr. Cabot: I have never seen a blood that contained nothing but polynuclears. X-ray treatment in blood diseases, especially leukemia, hits abnormal cells like the myelocyte very specifically, but in a case like this I do not know of any specific effects of x-ray treatment on the blood.

Differential Diagnosis

We have to find the cause of death of a man who was sick in this hospital for a month, who had been known to have enlarged glands for a very long time, but who had been acutely sick for only twelve days before he came here,—an illness of about forty days in all. The essential facts are (a) enlarged glands which go down under observation during the course of a fever; (b) a big spleen which also went down presumably for the same reason, whatever that is; (c) continuous ascites which did not change; (d) masses judged by the radiologist to be abnormal at the roots of the lungs; and (e) a secondary anemia. Now what can do that?

I do not know any variety of sepsis that can run that length of time with no acute lesion showing and no emboli or heart lesion to make us think it localized there. I speak of sepsis because it is the commonest cause of enlarged glands. I do not think it probable in this case. If we consider his enlarged glands, spleen and liver and ascites, sepsis will not account for it. Sepsis comes in, if at all, as a terminal complication.

Can we say syphilis? I think not. The glands alone and the spleen could be due to syphilis, but the course is not like any syphilis that I know of. People do not die of syphilis unless it hits some particular organ in a way that we have no evidence of here. The same can be said of tuberculosis.

I see no plausible diagnosis except malignant lymphoma or Hodgkin's disease: I mean a neoplasm starting in the lymph tissues in various parts of the body. Just where it is in the abdomen I do not know. We can easily suppose that it is in the root of the mesentery so as to impede circulation and produce ascites. It might be in the liver too, but as to that we can only speculate. Apparently it is in the root glands of the lungs. Now lymphoma has no characteristic blood. It has often been said that it has, but anybody who studies it will conclude that there is no characteristic blood of lymphoma. It often produces long periods of fever such as we have here, and in many cases we find an infection post mortem and think the fever due to that. The high percentage of polynuclears we expect in sepsis. Still "100 per cent" makes us think of something more than infection. I can hake no other diagnosis than lymphoma or lymphoblastoma (=Hodgkin's, malignant lymphoma, etc.)

A Student: Do you think the glands in childhood were due to the Hodgkin's?

Dr. Cabot: I do not know what else to think. But there are quite a number of cases on record in which glands have remained stationary and symptomless for a long time and then have begun to produce symptoms.

A Student: It there any record of basal metabolism?

Dr. Cabot: There is no record.

A Student: How about his abdominal masses?

Dr. Cabot: The pain which he had in the beginning sounded peristaltic. Of course if he had lymphoma it could perfectly well be in the intestine. It is one of the common places for lymphonma. Or even if it is outside the gut it can interfere enough to account for pain. If the pain is not from lymphomatous masses in relation to the intestine, I do not know what it is.

A Student: Could this be aleukemic leukemia?

Dr. Cabot: I should have said something about the bone marrow. We do not feel comfortable about making a diagnosis of neoplastic bone marrow involvement when we have 100 per cent polynuclears. That he should be putting out polynuclears with a marked involvement of the bone marrow would be very unusual. "Aleukemic leukemia" means myeloblastoma or lymphoblastoma of the marrow without any circulatory metastasis. I like those positive terms better than the negative one. I would rather state in our name for the disease the thing we know, which is the marrow lesions on the basis of their histology. I have given the chief points against that, namely, the presence of marrow cells and the exclusion of everything else in the blood.

A Student: Do you attach any importance to the fact that his children have enlarged glands?

Dr. Cabot: I do not know what importance to attach to it. Lymphoma is not hereditary so far as I know.

A Student: Can Gaucher's disease be ruled out?

Dr. Cabot: No, I do not know that it can. But the majority of cases have a much enlarged spleen.

A Student: How about gastric carcinoma, from the history of pain and the secondary anemia?

Dr. Cabot: I think that is rather little to support that diagnosis. We should have more evidence before thinking of gastric cancer, especially as he has so much else that can cause his secondary anemia.

A Student: Isn't the duration rather long for Hodgkin's?

Dr. Cabot: I don't think there is any duration that can be stated for Hodgkin's. I have known very long cases. I have known short ones.

A Student: What is the significance of the red-colored urine?

Dr. Cabot: Nothing, I think; high-colored, concentrated urine. It shows a gravity of 1.030 once. "Red" means high-colored. People use colors very loosely in talking about patients, as "green sickness" for chlorosis, that sickness which is never green. I think this is simply an exaggeration.

A Student: What were the lung symptoms at the end?

Dr. Cabot: "The rales increased in both sides of the chest, more so on the right." The respiratory rate rose. That is a good point to bring out. There may well be a bronchopneumonia. We do not need to suppose it, but it is quite possible.

A Student: Could sarcoma do this?

Dr. Cabot: You mean lymphosarcoma? You get out of my depth there. If you mean that it starts from the lymph glands themselves and especially from the immature cells, it seems to me lymphoblastoma is the better name.

A Student: How about the shooting pain in the leg?

Dr. Cabot: I don't know. I don't think that can be due to this disease.

A Student: How do you account for glands decreasing in size?

Dr. Cabot: X-ray treatment accounts for it. Perhaps also he had some terminal infection. Any terminal infection that we can name tends to make the glands and spleen decrease in size.

A Student: I was referring to the glands diminishing in his youth.

Dr. Cabot: I do not think I have anything to say about that.

A Student: What was the significance of the shiny stiff tongue?

Dr. Cabot: Fever, that is all.

A Student: Do not the glands in Hodgkin's disease recede at times?

Dr. Cabot: Yes, they do, especially under treatment.

A Student: How do you explain the tenderness of the inguinal glands?

Dr. Cabot: I do not explain it.

A Student: Doesn't the terminal blood picture of Hodgkin's disease reach 100 per cent polynuclears?

Dr. Cabot: I have never known it before.

Clinical Diagnosis (from Hospital Record)

Lymphoblastoma, Hodgkin's disease.

Terminal infection (bronchopneumonia?)

DR. TRACY B. MALLORY

Anatomic Diagnoses

Lymphoblastoma (Hodgkin's type.)

Bronchopneumonia.

The anatomical findings were those of a typical Hodgkin's disease. The lymph nodes throughout the body were enlarged, to notable size in the inguinal region and also in the root of the mesentery. There were tumor nodules scattered throughout the liver. The spleen was the typical "porphyry" spleen of this disease. There were innumerable nodules scattered throughout the lungs, some of them miliary in size, others reaching nearly a centimeter in diameter, also Hodgkin's disease, although in gross very closely simulating tuberculosis. The bone marrow was entirely negative.

There was a pneumonia in the right base, evidently present for a considerable time, since already a moderate degree of organization had occurred. Organization is of course a possible outcome of any pneumonia which fails to resolve, and although relatively uncommon we have seen several cases recently. The other organs were essentially negative except for acute tubular degeneration in the kidneys.

A Student: Was there any obstruction in the vena cava?

Dr. Mallory: I think perhaps, from the external pressure of these nodes.

A Student: Was the lung parenchyma involved?

Dr. Mallory: To a considerable extent. That is not very common, but it is quite possible. I have seen one case where there was excessive pulmonary involvement. The patient had been in a tuberculosis sanatorium for three years. He had also a vertebral lesion which was diagnosed by x-ray as Pott's disease. The x-ray pictures of the two conditions may be identical I believe.

A Student: Did you diagnose this disease from the biopsy material?

Dr. Mallory: Yes; it was a very typical Hodgkin's disease.

A Student: What other general disease can you diagnose from histologic sections of lymph nodes?

Dr. Mallory: Generalized tuberculosis of the lymph nodes. That is about all that I feel very sure of.

A Student: Do you use the term "aleukemic leukemia?"

Dr. Mallory: Not yet.

Dr. Cabot: In relation to this term, it doesn't make any great difference what we call things, but I think it is sensible to name a disease by what is there rather than by what is not there,—non-leukemic leukemia,—whereas we have something definite to say, namely, bone marrow growth—myeloblastoma.

CASE REPORT
(Liver Injury following Antiluetic Treatment.)

IRVING BANCROFT, M. D.,
Los Angeles, Calif.

A well developed and nourished girl, 21 years old in March. 1928, developed a typical primary sore on the vaginal mucous membrane, but never at any time had any secondary symptoms as treatment was begun soon after the appearance of the primary sore. Between March and November, 1928, she took 21 doses of Neosalvarsan, 6 grams at each dose and 21 doses of mercury salicylate, one grain at each dose. She presented no unusual reactions to the treatment except that after the first dose of each series, she felt sick. After the initial dose of the first two courses, she had general paines, backache and felt ill for a short time. After the first dose of the third course which was given after four weeks' vacation from all treatment, she became definitely sick with symptoms of anorexia, chills, fever, backache, nausea, epigastric pains and tenderness. In about three weeks she became jaundiced.

On December 11, 1928, she was still deeply jaundiced, nauseated and could eat little food. She was also definitely tender over the liver. The urine, at this time, contained no albumin, but much bile pigment. Sodium thiosulphate was given intravenously and sodium phosphate by mouth at this time, but she continued to grow worse.

On December 21, 1928, she entered the Los Angeles General Hospital. The examination at admission showed the heart and lungs to be negative, Wassermann negative, leucocytes 14,000, with 16 per cent lymphocytes and 84 per cent polymorphonuclears. The urine showed no albumen but there were a few hyaline casts and a few cells stained with bile. There was a definite tenderness over the right upper quadrant of the abdomen. The spleen was not palpable, the reflexes were normal. The blood coagulation time was 38 minutes, bleeding time 50 minutes. The urine functional test showed the appearance time to be 30 minutes and during the first half hour 45 per cent was excreted and during the second half hour 8 per cent was excreted.

On December 22. 1928, she was nauseated all the time and vomited blood at times. She had become deeply jaundiced. The blood chemistry at this time was as follows: non protein nitrogen 42 mg., uric acid 5 mg., calcium 8.2 mg. per 100 c.c. of blood. The icterus index was 40; initial hemolysis 36 per cent, complete hemolysis. 24 per cent. Death took place on December 31, 1928.

The autopsy (by Dr. Wagner) showed almost complete necrosis of the liver cells and with considerable infiltration of lymphoid and plasma cells. The kidney showed cloudy swelling and degeneration but no inflammatory infiltration.

There are several interesting points about this case.

1. The facts herein stated do not support the theory that hepatitis occurs only when the kidney does not function properly.
2. There is sometimes no warning of impending liver injury.
3. Injury occurred after the first dose which was given after four weeks vacation and hence the cumulative action of the remedies are hardly likely to be causable factors.

Southwestern Medicine

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JOINT SOCIETY AND HOSPITAL MEETINGS IN PHOENIX

The medical organizations in Maricopa County are attempting to solve the problem of too frequent medical meetings by combining the county society meetings with the hospital staff conferences. This type of combined meeting was discussed at the Conference of Secretaries of the American Medical Association at their meeting in November, 1928. In was subsequently proposed to the Maricopa County Medical Society and the staffs of St. Joseph's Hospital and the Good Samaritan Hospital, at Phoenix. The personnel of the three organizations being nearly identical, conditions seemed to be ideal for this arrangement. Having been approved by all organizations, the plan was inaugurated in January of this year and has, so far, been very successful. The attendance is better than ever before, and the programs have been very interesting. The former schedule of meetings included two meetings a month for the County Society and one meeting a month for each staff. The Society continues to meet semi-monthly, meeting with St. Joseph's Hospital Staff on the second Monday evening and with the Good Samaritan Hospital Staff on the fourth Monday evening. One organization occupies the first hour; then adjourning while the second organization's officers take charge and hold their meeting; the personnel of the two organizations being the same, the only interruption being to change the presiding officers. The Staff Conference follows the recognized procedure of discussing cases treated in the hospital, while the Society portion of the meeting usually includes papers or address by some invited guest. This diversity of program, with the semi-monthly instead of weekly meetings, has served to increase the interest and attendance.

CHANGE DATES FOR ARIZONA STATE MEETING

After the dates of the Arizona State Medical Association meeting had been set for April 17, 18 and 19, and the Public Health Association meeting for April 15 and 16, objection was voiced to these dates as coming immediately preceding Easter. The Program Committee decided to change the dates to a week later, and were fortunate in securing the cooperation of all invited guests for the change in dates. The Public Health Association also changed their dates to April 22 and 23. The full program of both these meetings will be found elsewhere in this issue.

CHARLES H. SAWYER

Dr. Charles H. Sawyer, for many years in the Indian Service, with location at San Carlos, and of recent years practicing in Globe, Ariz., died on Februaryl 22nd. He was a member of the Gila County Medical Society, the Arizona State Medical Association and a Fellow of the American Medical Association.

Dr. Sawyer was born in 1857, and graduated from the Western Reserve University at Cleveland in 1880.

EASTMAN MEDICAL FILMS:—The American College of Surgeons is sponsoring the production of medical and surgical pictures, the technical work being done by the Eastman Kodak Company. These films can be either rented or purchased from the Eastman Teaching Films, Inc., Rochester. N. Y. They are made in two sizes,—the 16 mm. film which is the size for use with the Eastman projector, and the standard movie film of 35 mm.

NEW SOUTHERN PACIFIC HOSPITAL IN TUCSON:- Dr. W. B. Coffey, Chief Surgeon of the Southern Pacific, visited Tucson recently in the interest of the proposed new hospital to be built there by the Southern Pacific Railroad. This will be a notable addition to the group of industrial hospitals in Arizona, and to the hospital facilities in Tucson. When completed, it will place Tucson in the forefront of the cities of the Southwest, so far as hospital facilities are concerned.

The Arizona State Medical Association
THIRTY NINTH ANNUAL MEETING
Phoenix, Arizona, April 24, 25 and 26

Headquarters—Hotel Westward Ho

ANNOUNCEMENTS

Registration headquarters and all scientific and business meetings will be held in the Hotel Westward Ho. Every member, visitor and guest is requested to register promptly on arrival.

Scientific sessions will be held in the ballroom of the hotel and will begin promptly on time.

No address or paper before the Association, except those of invited guests, shall occupy more than twenty minutes. The opening discussions are limited to five minutes and general discussions to three minutes each. No one shall speak more than twice on the same subject.

Papers read before the scientific sessions shall become the property of the Association and shall be deposited with the secretary for publication in the official organ of the Association (Southwestern Medicine.)

Special announcements will be sent out about the social entertainments.

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PROGRAM OF SCIENTIFIC SESSIONS

THURSDAY, 10:00 A. M.

Address of Welcome:
HON. JOHN C. PHILLIPS, Governor of Arizona.
Introduction of President-Elect.
President's Address:
JOSEPH M. GREER, Phoenix.
C. C. TIFFIN - - - - - - - - Seattle, Wash.
"Surgical Treatment of Goitre."
Discussion opened by
E. PAYNE PALMER, Phoenix.

Symposium on Industrial Medicine and Surgery

JOHN E. BACON and WM. B. WATTS, Jr. - Miami
"Arthritis and Its Relation to Industrial Medicine."
JOHN W. FLINN - - - - - - - - - Prescott
"Pneumoconiosis and Silicosis."
A. C. KINGSLEY - - - - - - - - - Phoenix
"Back Injuries."
CHAS. N. PLOUSSARD - - - - - - - - Phoenix
"Inflictions of the Fingers and Hand."
WM. LYLE BELL - - - - - - - Oakland, Cal.
"Organic Internal Splinting of the Fractured or Detached Olecranon."
JOHN TAHENEY - - - - - - - - - Phoenix
"The Relation of the Medical Fraternity to the State Industrial Commission."
Discussion on industrial papers opened by
JAMES R. MOORE, Jerome.
R. D. KENNEDY, Globe.
S. H. WATSON, Tucson.
GEORGE A. BRIDGE, Phoenix.

Symposium on Chest

FRIDAY, 10:00 A. M.

ALLAN K. KRAUSE, Director, Desert Sanitarium, Tucson.
"Trends in Treatment of Pulmonary Tuberculosis."
C. S. KIBLER and S. H. WATSON - - - Tucson
"Management of Artificial Pneumothorax Patient."
J. L. MCKNIGHT, U. S. Veterans' Hospital. Tucson
"Intestinal Tuberculosis from the Standpoint of the Roentgenologist."
W. R. LEVERTON - - - - - - - - - Tucson
"Some Phases of the Heart in Tuberculosis and Other Lung Diseases Including Studies with the Electrocardiogram."
VICTOR RANDOLPH and W. W. WATKINS - Phoenix
"Limitations and Correlated Values of Clinical and X-ray Findings in Chest Diagnosis."
Discussion of papers opened by
E. W. PHILLIPS, Phoenix.
VICTOR RANDOLPH, Phoenix.
FRED G. HOLMES, Phoenix.
H. L. GOSS, Phoenix,
O. C. WEST - - - - - - - - - - Phoenix
"Epidemiological Studies."
C. E. YOUNT - - - - - - - - - - Prescott
"Hydatiform Mole, with Case Report."
Discussion opened by
FLOYD B. SHARP. Phoenix.

SATURDAY, 10:00 A. M.

CHAS. T. STURGEON - - - - - - Los Angeles
"Pulsion Diverticulum of the Esophagus."
F. M. POTTENGER - - - - - Monrovia, Cal.
"The Explanation of Symptoms."
HARRY A. REESE - - - - - - - - - Yuma
"Fever."
Discussion opened by
ROBERT S. FLINN, Prescott.
HARRY T. SOUTHWORTH - - - - - - Prescott
"Ovarian Hematomas of the Endometrial Type (Chocolate Cysts), with Report of Cases."
Discussion opened by
GEORGE E. GOODRICH, Phoenix.
C. C. TIFFIN - - - - - - - - Seattle, Wash.
"Differential Diagnosis and the Medical Treatment of Goitre."
Discussion opened by
S. I. BLOOMHARDT, Phoenix.
A. C. CARLSON. - - - - - - - - - Jerome
"Volkmann's Ischemic Contracture." (With report of Case and Moving Pictures).
Discussion opened by
EDGAR BROWN, Phoenix, Arizona.

ARIZONA PUBLIC HEALTH ASSOCIATION

The third annual meeting of the Arizona Public Health Association will be held at the Adams Hotel, Phoenix, Arizona, on Tuesday and Wednesday, April 22 and 23, under the auspices of the State Board of Health. Health officers, city engineers, water works and sewage plant operators, dairy inspectors, public health nurses, and all others actively interested in community health and sanitation are cordially invited to attend this meeting.

Tuesday, April 22
9:00 a. m.
GENERAL MEETING
H. T. Southworth, presiding

Registration and Organization Preliminaries.
9:30 a. m.: Address of Welcome—F. J. Paddock.
Local arrangements for meeting—Phoenix Committee.
Introduction of Registrants.
State Board of Health Program—R. J. Stroud, M.D.
Organized Health Administration in Texas — J. C. Anderson, M.D.
Moving Pictures of Creamery Waste Treatment — Wisconsin Division of Sanitary Engineering. Courtesy of L. F. Warrick.
Chlorination for Odor Control at Mission Creamery——James Minotto.
Discussion—G. T. Luippold and N. T. Veatch, Jr.
10:30 A.M. Visit to Mission Creamery and Waste Disposal Field.

Tuesday, April 22
2:00 p. m.
Joint Meeting of Health Officers and and Dairy Inspectors' Sections.
Dairying in Southern California—Jay Dutter.
Dairying in Foreign Lands—C. O. Hagen.
The Present Status of the Dairy Industry in Sonora Dairy Commissioner of Sonora.
Enforcement of Dairy Regulations in Utah—Edward Southwick.
Dairy Conditions in Northern California—Sam H. Greene.
Pasteurization—George W. Putnam.

WATER AND SEWAGE
2:00 p. m.
N. T. Veatch, Jr., presiding

Planning and Zoning their Relationship to Sanitation—W. J. Jamieson.
Selection of Deep Well Turbine Pumps—Richard Bennett. Discussion—H. Schwalen, Roland Taylor, and F. R. MacPherson.
Superior Water Works Improvements—M. A.Moody.
Method of Maintenance Used by Phoenix Water Department—R. T. Gardner. Discussion —F. C. Lynes and A. J. Kline.
Controlling Algae Growth at Winslow—R. C. Kline. Discussion—Richard Bennett and Jane H. Ryder.
Swimming Pool Sanitation—G. T. Luippold. Discussion—D. W. Swihart.
Classification of Algae Found in Reservoirs—H. L. Shantz.

EVENING
7:00 p. m. Dinner—H. T. Southworth, M.D., presiding.
Public Health Organizations—Homer N. Calver.
Talk on Health Policies—Honorable John C. Phillips.
Annual Meeting of Arizona Public Health Association.

Wednesday, April 23
9:00 a. m.
HEALTH OFFICERS' SECTION
F. T. Foard, M.D., presiding

Diphtheria Toxin-antitoxin — O. C. Wescot, M. D. Discussion—R. B. Durfee, M.D.
Health Work in Pima County Rural Schools—Helen Bocock, R. N.
Health Work in the El Paso Public and Private Schools—P. R. Outlaw, M. D.
Methods of Cooperation Between Individual Counties and the State Health Nurses—Anna H. Osback, R. N.
Control of Meningitis in Gila County—C. E. Irvin, M.D. Discussion—J. W. Morris, M.D., N. D. Brayton, M.D., and H. A. Spencer, M.D., U.S.P.H.S.
The Benefit of the County Health Unit—V. G. Presson, M.D.

DAIRY INSPECTORS' SECTION
T. P. Morgan, presiding

The Success of the Standard Milk Ordinance in Tucson—J. C. Hicks, D.V.M.
Progress of Standard Milk Ordinance—Milk Specialist, U.S.P.H.S.
The Dairy Industry in Yuma County— Homer T. Kryger.
Certified Milk—J. P. Bushon, M.D.
Enforcement of Standard Ordinance, Section by Section.

WATER AND SEWAGE SECTION
Chester A. Smith, presiding

Construction of Sewers—Chester A. Smith. Discussion—George Grove.
Disposal of Sewage from Institutions—Chester Cohen. Discussion—M. R. Tillotson and L. J. Mahoney.
Aeration of Sewage—N. T. Veatch Jr.
Indianapolis Sewage Treatment Plant—Dario Travaini.
Vacuum Degassification and Circulation of Sludge—S. A. Adams. Discussion—Chester Cohen.
Construction and Operation of Small Sewage Treatment Plants. —H. B. Hommon.
Informal luncheon for Water and Sewage Section—Adams Hotel.

Wednesday, April 23
2:30 p. m.
HEALTH OFFICERS' SECTION
R. J. Stroud, M. D., presiding

Round Table Discussion of Public Health Problems in Arizona.
Incidence of Dysentery in the Salt River Valley—Harry Carson, M.D.
Communicable Disease Control and Quarantine in Arizona.—O. C. West, M.D.

DAIRY INSPECTORS' SECTION

Field Trip to Dairies and Creameries.
This trip will include a visit to raw and pasteurized milk plants, an ice cream factory, and condensery.

WATER AND SEWAGE SECTION
H. B. Hommon, presiding

Round Table Discussion.
Sterilization of Water Mains—G. T. Luippold and A. J. Kline.
Operation of Sewage Treatment Plants—D. Travaini. Discussion—Hillyard Hall.
Garbage Collection and Disposal—M. R. Tillotson. Discussion—Chester A. Smith and Chester Cohen.
6:30 p. m. Dinner—R. N. Looney, M.D., presiding.
Undulant Fever—J. C. Geiger, M.D., speaker.

The Southern Pacific Health Exhibition car will be in Phoenix during the meeting. Definite arrangement for the time of their demonstration will be made later. This car has been sent to Phoenix through the courtesy of Dr. W. B. Coffey, Chief Surgeon for the Southern Pacific Company.

SPECIFIC THERAPY OF ERYSIPELAS

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PERSONALS

LOCATION FOR DOCTOR IN NEW MEXICO:—Some of the citizens of San Jon in New Mexico are interesting in securing a physician for that location. This is a farming country and San Jon is the center of the district which is said to be forty miles or more square. Any doctor interested should write Mrs. J. D. Griffiths, San Jon, N. M.

DR. M. B. CIRLIN, of Chicago, Ill., has been visiting friends in Phoenix during February.

DR. FRANKLIN H. MARTIN, of Chicago, Director General of the American College of Surgeons, who is spending several weeks at the Biltmore Hotel in Phoenix, recently entertained Arizona members of the College at a dinner. Most of the College fellows were present, coming from all parts of the state.

DRS. J. J. BEATTY AND W. R. LEVERTON, of Tucson, announce the opening of offices in the Consolidated National Bank Building, with practice limited to the diagnosis and treatment of the heart and lungs including x-ray and electrocardiographic studies.

DR. J. C. GEIGER, of the United States Public Health Service, San Francisco, was a visitor in Maricopa County, early in March. He will be in this vicinity frequently during the spring, as the Public Health Service is conducting an extensive investigation into malta fever and abortus infections.

EL PASO COUNTY MEDICAL SOCIETY

Meeting Feb. 10, 1930

Meeting called to order by the president, Dr. Paul Gallagher, at 8 p. m. There were 50 members and 5 visitors present.

The minutes of the previous meeting were read and approved.

The president explained to the Society that the instructions received at the previous meeting which provided for paid advertisements in all three local papers relative to Periodic Health Examinations, as well as news insertion of a synopsis of that meeting had not been carried out by the officers of the Society because, after consultation with advertising experts, it was deemed that the cost of such advertisements would be out of proportion to the benefit derived. Dr. Waite then made a motion, seconded by Dr. R. B. Homan, that the whole matter be dropped. Motion carried.

The paper of the evening was read by DR. GEORGE TURNER. It was entitled "Preliminary Report of Experimental Studies in Deprotinizing Blood Serums." He stated that the chemical method of precipitating protein from solution by the use of salts of heavy metals and by the use of alkaloidal reagents is a familiar study in biochemistry. His method, apparently untried before, consists of the precipitation of the protein by electrolysis. Pure gold as the anodal plate is the only metal with which he has been successful so far. A platinum plate is used as the cathode. The passage of a weak direct current through the serum causes a

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HEADQUARTERS---

for the

Convention

of the Arizona State Medical Association

at Phoenix
April 24, 25, 26

at

Westward Ho

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Special rates are available to the members in attendance at this convention.

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precipitation of its protein, both primary and secondary, either collecting at the bottom of the cell or tube, or accumulatnig on the anode, depending on whether the serum is diluted or concentrated. The remaining protein free solution is slightly alkaline. By rendering it slightly acid, it is possible to precipitate the antitoxic element which can then be redissolved in a minimum amount of very slightly alkalinized physiological salt solution. The practical applications and the conclusions at which he arrived are:

1. The deproteinizing of blood serum by electrolysis is practical and not difficult.
2. It separates and removes the total protein from the toxic or antitoxic elements of the serum.
3. The antitoxic elements act in their specific manner more precisely after the protein is removed.
4. The antitoxic element with the protein removed keeps better and tolerates a higher temperature.
5. The removal of the protein from the serum by electrolysis makes the complement fixation test more reliable.
6. It makes the laboratory detection of tuberculosis possible from the blood serum.
7. It removes the objectionable and, at times, dangerous element from antitoxic sera.

In the discussion, DR. STEPHEN SCHUSTER asked how much activity must be present to be able to detect tuberculosis from tests of the blood serum. He also asked if this test would give any idea as to the location and amount of involvement.

DR. LEIGH thought Dr. Turner was deserving of a great deal of credit for his work. It recalled to his mind the work done in the isolation of insulin. He thought that with this method it might ultimately be possible to permit the availability of antitoxin in a dry form and thus extend its use to more remote localities.

DR. RALPH HOMAN thought that this test would be of special benefit in the early diagnosis of tuberculosis. He cited one case which Dr. Turner reported positive when clinically it was thought to be negative for tuberculosis. Subsequent developments proved Dr. Turner to be right.

DR. LAWS complimented Dr. Turner and stated that the extent of the practical applications of his experiments could not be fully realized as yet. As to the complement fixation test in tuberculosis, if it turned out as satisfactory as Dr. Turner thought it would, it would be revolutionary, as heretofore it had not been of much practical value.

DR. RIGNEY thought that the practical applications of these experiments would be as beneficial from a therapeutic standpoint as from a diagnostic one, as the removal of the protein from serums would remove the danger of anaphylaxis and serum sickness.

DR. TURNER, in closing, stated that he hoped and thought that this method of deproteinizing serum would prove beneficial both from a therapeutic and a diagnostic standpoint. He pointed out that the harmful reactions now occasionally met with in the administration of antitoxins are not produced by the antitoxin element, but by the protein in the serum. In answering Dr. Schuster, he stated that he had tested the serum from a number of patients in Dr. R. B. Homan's Sanatorium, both tuberculous and non-tuberculous, and that all cases that had a temperature from tuberculosis were indicated by the test and vice versa. That one case that was thought to be tuberculous, clinically, but had a negative test, went on to autopsy and was proven non-tubercuous. As to the location of the lesion, this could not be

DOCTOR

The

Dallas Southern Clinical Society

Will Hold the Next Meeting

In Dallas, April 14, 15, 16, 17, and 18. There will be Lectures, Clinics and Post Graduate Work, covering Medicine, Surgery and the Specialties—this will be done in the forenoon.

Each afternoon will be devoted to General Meetings when men of national reputation will address you. Motion pictures will be shown, among them being the famous Canti (Cancer) film.

Remember the date, April 14-15-16-17-18
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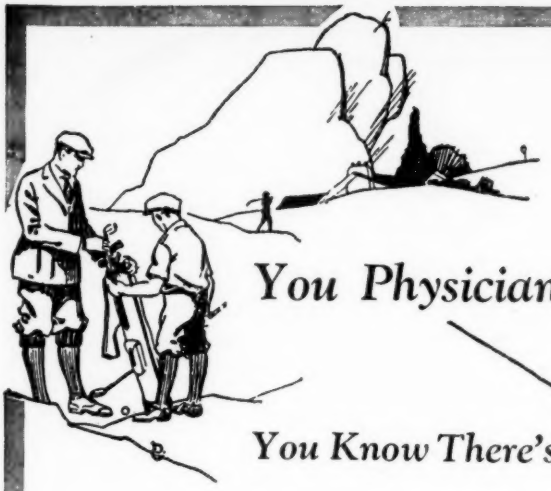

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You Know There's a Club for Every Stroke

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Similarly in artificial infant feeding. For the normal infant, you prefer cow's milk dilutions. For the athreptic or vomiting baby, you choose lactic acid milk. When there is diarrhea or marasmus, you decide upon protein milk. In certain other situations, your judgment is evaporated milk.

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told, and made no difference as far as the test itself was concerned. The hydropenion of the electrolyte is important, for if you swing it to acid, the whole thing will spoil. What the precipitate of antitoxin on the anode consists of is not known, but once the antitoxin is precipitated it loses its properties. He expected to do a lot more work on these experiments.

DR. J. J. GORMAN reported a case of pernicious anemia, detailed account of which will be found published elsewhere in this journal (April issue.)

MR. HOGAN, of the El Paso Times, addressed the Society in regard to a special edition of their paper to appear shortly. He said a committee (apparently himself or his newspaper) were selecting prominent citizens, past, present and future, and inviting them to have their photographs printed in this edition. He assured us that the motive was entirely altruistic and that only those invited would be permitted the privilege of seeing their resemblances that coming morning in print. He wanted the County Society to sanction the participation of those of its members who might be invited. After he had left Dr. Ralph Homan inquired as to what the charges would be for such a privilege. The President informed him that it was $20.00 per. Dr. Homan then made a motion that Mr. Hodge's proposal be not accepted. Seconded and carried.

The President then informed the Society that the U.S.P.H Service requested that this body endorse House Bill No. 3142 and Senate Bill No. 1195, which is "To Provide for Coordination of Public Health Activities of the Government." Dr. Waite made a motion that this bill be endorsed and that the Secretary be instructed to communicate this endorsement to our Senators and Representatives. Motion seconded and carried.

Dr. Gorman requested that two copies of all papers intended to be read before the State Association must first be presented to the County Society, and that members having such papers get in touch with Dr. Ralph Homan promptly so that they might get on the program early. He called attention to the fact that about forty-seven members have not yet paid their dues and requested that they do so. He announced that dinner would be served at 7 p. m. at Hotel Hussmann on meeting nights to those members who cared to participate. At these dinners matters to be brought up at the meeting could be discussed and thus the business of the meeting expedited.

There being no further business the meeting adjourned at 9:15 p.m.

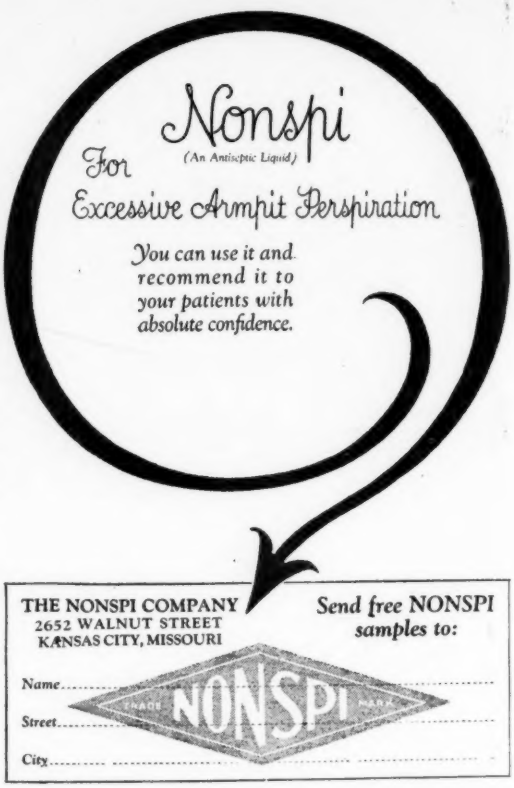
S. H. NEWMAN, M.D.,
Secretary.

February 24, 1930

Meeting called to order by the president, Dr. Paul Gallagher, at 8 p.m. There were 51 members and two visitors present.

On motion made by Dr. Waite, the reading of the minutes of the previous meeting were dispensed with.

The first paper of the evening was that of DR. F. C. GOODWIN, entitled "Some Foot Problems," In it he pointed out that the weak painful foot is the most common postural condition which is referred to the office. From the history you are usually able to rule out infection. That due to malalignment is characterized by pain in the heels, apex of the inner long arch behind each malleolus with stiffness in the calf muscles. The foot is usually abducted, producing what seems to be a flat foot, but the foot is not flat. The arch can be completely restored if the foot is put in proper alignment. Overweight, long hours of standing, a bunion,



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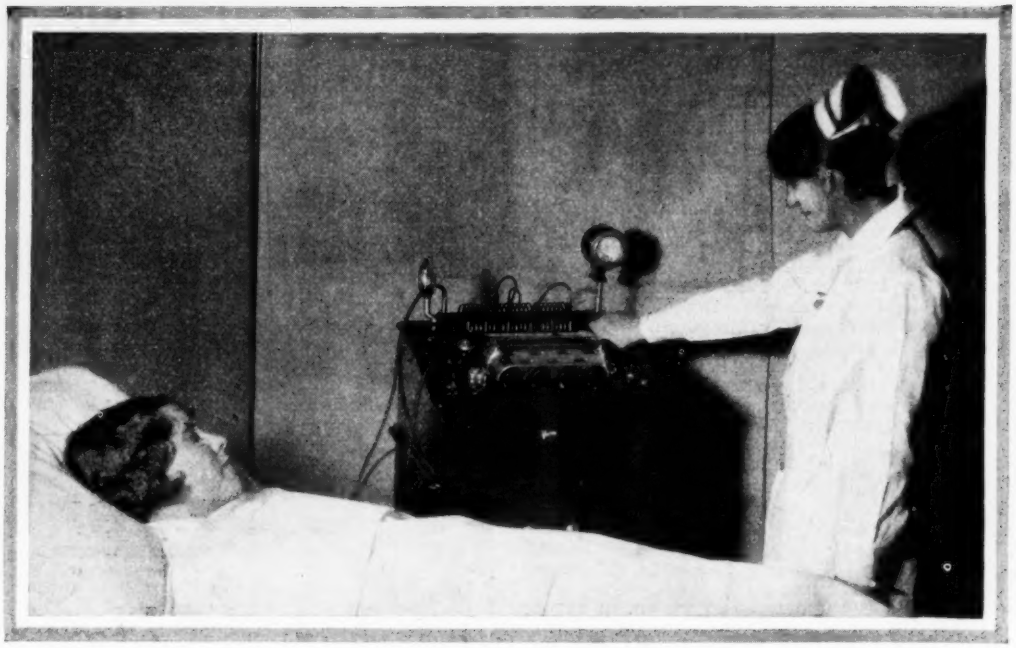

An Effective Ally in the Treatment of Pneumonia

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In the above illustration the apparatus proper is shown mounted on a floor cabinet, from which it may be lifted and conveniently taken in your auto to the patient's home.

A REPORT from the Department of Physiotherapy of a well-known New York hospital, dealing with diathermy in pneumonia and its sequelae, states as follows:

"As a rule diathermy is indicated in acute pneumonia, especially so when the symptoms are becoming or already are alarming: the temperature is high, the patient is delirious, the pulse is extremely rapid, cyanosis is deep, the respiration rate is high, the breathing is very shallow, and the cough remains unproductive. Not infrequently in a pneumonia case with such alarming symptoms, after a few diathermy treatments an entire change of the picture takes place: cyanosis lessens, respiration becomes deeper, the quality of pulse improves, the rate decreases, the temperature is lowered, and the cough becomes productive. Auricular fibrillation that develops occasionally in similar pneumonias or other types of pneumonia where the toxemia is great, has been changed to a perfect normal rhythm after a few diathermy treatments."

You will value diathermy as an ally in your battles with pneumonia at this season, aside from the satisfaction derived from having utilized every proved therapeutic measure that present day medical science offers.

A reprint in full of the article above quoted, also reprints of other articles on this subject, will be sent on request.

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deformed toe, or anything which causes the patient to favor one foot or to overwork any group of muscles may be the causative factor. In these cases the first appearance of pain in the foot is at the attachment of the plantar ligament to the os calcis. Then the pain soon begins to appear at the astragalonavicular joint. Then pain soon appears posterior to each malleolus. Pain may appear in the knee joint due to torsion of the tibia and in turn the femur is rotated inward, causing tension on the psoas major muscle which in turn pulls on the lumbar vertebrae, producing low back pain. In the treatment of these conditions, the feet should be strapped with large felt pads, pulling the foot into the corrected position. Repeat once a week. Keep patient active. Use large loose shoe with Thomas heel, which may or may not be elevated. If not markedly improved in three weeks, then a focus of infection should be sought for and crorected if found. Such cases as complain of pain in the course of the vessels to the sole of the foot between the os calcis and medial malleolus.

For the correction of bunions, the Mayo operation should not be performed, as it is a destructive operation and the results are often pitiful. The Silver operation should be the one of choice, as it is corrective and not destructive.

Metatarsalgia is encountered in women in about 95 per cent of the cases. The cause is high heel and tight-fitting, pointed shoe, squeezing the ends of the metatarsals together. To relieve, place a large support or elevation far up under the heads of the metatarsals.

In cases of calcaneovalgus, supply the child with flat, rigid shoes, with the inner side of the heel elevated an eighth of an inch. A large felt pad is applied to the shoe so as to support the inner long arch. A few may need plaster cast.

Fracture of the os calcis with displacement, invariably causes persistent pain and disability. The treatment is triple arthrodesis.

The second paper of the evening was entitled "Public Health with Especial Reference to its Problems and Administration," by SURGEON J. R. HURLEY of the United States Public Health Service. Public health considers both the person and his environment and resolves itself into hygiene and sanitation. It is a purchasable commodity but its cost is so high that public opinion and our legislative bodies are unwilling or unable to provide the price. From fifty cents to a dollar per capita is required for efficient local health administration. Modern civilization is what has rendered public health problems so complex. Four prime considerations are required to produce and insure good public health. These are, an ample supply of safely potable water; adequate, properly designed sewage disposal system; an adequate and efficient system for the disposal of garbage; and a properly organized city health department. Supplementing this, there should be a proper county or district health organization and also a state health organization, assisted when need be by the power of the Federal Government. Man incurs his diseases by direct contact with other persons, from the lower animals, from insects, through foods, through the water, and from the air. Laws or ordinances must be enacted recognizing these dangers to the public health and authorizing the organization of health departments to control them. These laws must also provide certain standards, must provide personnel and money. In most cities the Board of Health is invested with the responsibility of protecting the public health and supervising the health department. The latter should have a full time executive officer, who should be a physician skilled in preventive medicine and hygiene, and should comprise the following divisions, each with a competent chief in charge: A Division of Communicable Diseases; a Division of Child Hygiene; a Division of Sanitation; a Food Division; a Laboratory Dvision; a Division of Vital Statistics, and a Division of Publicity and Public Health Organization.

The County or District Health Organizations are not usually so extensive as those existing in larger cities and do not overlap or duplicate the work of the City Health Department. It should be headed by a full time physician.

The State Health Department is organized along lines similar to those described for the municipality. It acts in a supervisory and advisory manner, and, in addition, looks out for large-scale nuisances.

The Federal Government maintains the United

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Dr. Mary L. Neff, Trinity Hotel

Dr. Samuel D. Ingham, Roosevelt Building

States Public Health Service to handle problems too large for the States to cope with. It was organized in 1798 and has been twice reorganized. It is presided over by a Surgeon General with a commissioned medical corps and other special experts. Its duties are the protection of the United States from the introduction of disease from without and the prevention of the spread of disease in interstate traffic; the suppression of large epidemics; the investigation of those diseases affecting man, and the dissemination of health information. It also supervises as to purity and potency the manufacture of biological products.

Public health nursing is of great importance in public health work. This movement is of comparatively recent origin, but it has been growing steadily and has now received general recognition and is an established and important part of public health work. For a city of 100,000 inhabitants, the following nurses are required:

1 nurse for communicable disease control;
4 nurses for tuberculosis control;
2 nurses for venereal disease control;
15 nurses for infant welfare work;
8 nurses for school hygiene work. Total of 30 nurses.

The need for sufficient public health nurses is an outstanding problem. There should be upward of 50,000 public health nurses to serve the population of the United States instead of the approximatetly 12,000 to 15,000 in the field at present. Special schools or special courses in the nurses training schools are absolutely requisite.

DR. McNEIL, in commenting on Dr. Hurley's paper, stated that this was the kind of paper that was too infrequently brought before this Society. We do not always have a clear conception of the duties of the various health organizations and these matters could be clarified by the more frequent presentation of similar papers.

Dr. A. W. Multhauf, who was to have read a paper on "Prostatic Calculi" was unable to be present on account of sickness.

Our president announced that he was in receipt of an anonymous communication relative to advertising by some of our members. He wished it clearly understood that all anonymous letters received by him would be relegated to the wastepaper basket.

There being no further business, the meeting adjourned at 8:45 p.m.

S. H. NEWMAN, M. D.,
Secretary.

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CLEVELAND CLINIC FOUNDATION FELLOWSHIPS

The Cleveland Clinic Foundation offers four two-year Fellowships in Medicine beginning on July 15th of each year. to candidates who have had at least one year of hospital intern service. Appointments for the second year depend upon the character of service rendered during the first year and are subject to mutual agreement between the Directors and each Fellow. These Fellowships carry a payment of $1,200 for the first and $1,500 for the second year and give the holder the opportunity for an extensive and varied practical service.

A course of Fellowship Lectures extending over two years is given on each Monday evening from September to May inclusive.

A review of the x-ray films of current cases is given one evening each week during the above nine months and the Fellows are expected also to attend the weekly Pathological Conferences and the regular weekly meetings of the Medical Staff, at which various clinical problems are discussed.

Application blanks will be forwarded upon request to the Secretary of the Fellowship Committee, Cleveland Clinic, Cleveland, Ohio.

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